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INVESTIGATIONS

INTO SOME

MORBID CARDIAC CONDITIONS,

INCLUDING THE "CARTWRIGHT" PRIZE ESSAY ON

"THE HEART IN DEBILITY"

BY

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ILLUSTRATED WITH WOODCUTS, TRACINGS AND TEMPERATURE CHARTS.

EDINBURGH:
BELL & BRADFUTE, 12 BANK STREET.
LONDON: SIMPKIN, MARSHALL & CO., AND
HAMILTON, ADAMS & CO.
1886.

EDINBURGH
PRINTED BY LORIMER AND GILLIES,
31 ST. ANDREW SQUARE.

TO

G. W. BALFOUR, M.D., LL.D.,

EMERITUS PRESIDENT OF THE ROYAL COLLEGE OF PHYSICIANS OF EDINBURGH,

IN RECOGNITION OF HIS VALUABLE CONTRIBUTIONS TO THE STUDY OF CARDIAC DISEASE,

AND AS

AN ACKNOWLEDGMENT OF PERSONAL INDEBTEDNESS TO HIS TEACHING.

PREFACE.

THE greater part of these pages was presented as a Thesis for the Doctor of Medicine of Edinburgh, and was awarded a Gold Medal by the University. Chapters II. and III., under the title of "The Heart in Debility," was this year awarded the "Cartwright Prize," of five hundred dollars, by the Alumni Association of the College of Physicians and Surgeons of New York.

It is imperative on the successful competitor for the latter to publish his essay. This necessity, coupled with the representations of those for whose opinion I have considerable respect, has led me to publish these investigations in book form.

No one can be more conscious than I of their imperfections and incompleteness, but from the favour with which they have already been received, I venture to hope they may, to some extent at least, commend themselves to a wider tribunal.

I have to express my obligations to my friends Dr. Gibson and Mr. Cowper, for their kindness in willingly bestowing time and labour on the revision of the proofs, by which means many mistakes, which might otherwise have escaped detection, have been removed.

W. R.

EDINBURGH, *November, 1885.*

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INVESTIGATIONS

INTO SOME

MORBID CARDIAC CONDITIONS.

CHAPTER I.

HISTORICAL INQUIRY CONCERNING INORGANIC MURMURS, ETC.

IN such an inquiry as is here undertaken, it is not only necessary to state the opinions and teaching of living authorities, but also to note the birth and trace the evolution of these opinions. This last is fraught with interest. It shows not only how truth but also how error originated; how the authoritative and beneficial teaching of one age may mark a great advance on the knowledge of preceding ages, and yet act as a barrier to further progress in succeeding time.

Until the discovery of the circulation of the blood by Harvey, the diseases of the vascular system belonged to the vast *terra incognita* of early medicine. With this discovery, knowledge and comprehension of the various derangements of the system grew surely though slowly. Symptoms and the more obvious signs were long the only finger-posts pointing towards diagnosis; but to the man of highly developed powers of observation and of long experience, even these acquired a diagnostic and a prog-

nostic value which, to the man made skilful only by the precision of modern methods, it is difficult, if not impossible, to comprehend.

The pulse was studied, and its state revealed much to the experienced. Palpation, by applying the hand to the chest, was also practised. Auenbrugger published in 1761 a work on percussion ("Inventum novum ex Percussione thoracis humani, ut signo, abstrusor interni pectoris mortis detegendi"); which, however, only became widely known when translated by Corvisart and published more than forty years afterwards.

It fell to the lot of Laennec to make the next great advance. In 1818 he made a communication to the Academy of Sciences on Mediate Auscultation, and in the following year published the first edition of his work, "De l'Auscultation Mediate," &c. Others had listened to the sounds produced within the chest, and probably even acquired thereby some knowledge of the condition of the organs within; but it was the genius and industry of Laennec which gave to the profession a system to which certainly much has been added, especially as regards the circulation, and which, while containing grave errors, was a huge advance on the previously existing methods of investigation by inspection, palpation and percussion.

Laennec showed that the two methods of inspection and palpation were often misleading, or of negative value, since they were influenced by the physical conditions of the chest-wall, as in obesity and œdema. Palpation was regarded by him as of less value than the radial pulse for testing the regularity or irregularity of the heart's action.

He divided the precordia into two regions. The *first* he placed over the lower third of the sternum: it corresponded with the right side of the heart, and the sounds

heard here were referred to the right heart. The *second* he placed over the cartilages of the fourth, fifth, sixth and seventh sternal cartilages, and corresponded with the left heart. Here its motions were mainly perceptible, and the sounds heard here were referred to its contractions ("A Treatise on Diseases of the Chest," &c., Forbes' translation, 4th edition, p. 494 *et seq.*). The first sound of the heart he attributed to the ventricular systole, and the second to the auricular systole. Localising the sounds of the two sides of the heart to the respective regions stated above, led to unfortunate error, the influence of which is discernible in some of the teaching of the present time.

He held that in diseases of the heart "the pulse was often feeble, sometimes even imperceptible, although the heart's contraction, that especially of the left ventricle, was much more energetic than natural." From the position he selected for examining the left heart, he could not have failed to fall into this error. It further led him to discredit, almost wholly, information afforded by the pulse. In hypertrophy he believed the pulse to be as frequently weak as strong. While Corvisart recognised the soft and weak pulse as an indication of a dilated left ventricle, Laennec repudiated it, and considered "the clear and sonorous contractions of the heart between the fifth and seventh ribs as the only sure indication" (p. 554). This "sonorous contraction" over the lower part of the sternum, was in a similar way held to indicate dilatation of the right ventricle; but he also accepted the jugular pulse or jugular distension, the observation of which he ascribed to Lancisi, as a valuable sign of the condition.

Laennec was the first to describe the murmurs produced within the circulatory apparatus. He recognised a bellows

sound over the heart synchronous with the first or second sounds, or, according to his view, with ventricular or auricular contraction. He recognised a similar bellows sound in the arteries; and described a continuous murmur, "like that of the sea, or that which is produced by the application of a large shell to the ear" (p. 517). This is evidently the *bruit de diable* well known to modern observers. The bellows sound was found by him in hypochondriacs, cachectic persons, and those subject to hæmorrhages, as well as in cases exhibiting endocardial changes of a structural nature. This naturally led to confusion, the outcome of which was, that he believed murmur was often due to spasmodic contraction of the heart or arteries. This brief outline of the position of the father of auscultation cannot in the present inquiry be completed without referring to his belief, that softening of the walls of the heart was one of the causes which render the sounds of the heart more obtuse than natural, and he asks: "Could it account for that frequency of pulse which exists, sometimes for several weeks, in convalescence from fevers, although the patient continues to regain flesh and vigour" (p. 566 *et seq.*)?

Adams (*Dublin Hospital Reports*, vol. iv., 1827), in several interesting cases of stenosis of the left auriculo-ventricular orifice, noted the small weak pulse associated with forcible and extensive cardiac impulse, the impulses being more numerous than the radial pulse. He also noted the distension and pulsation of the cervical veins. In his review of these cases he argues against the views then generally held, and submits that the extended and increased cardiac impulse is due to the enlarged right heart, which completely covers the diminished left ventricle, as he repeatedly had proved by *post-mortem* examin-

ation. He found the jugular pulsation to be synchronous with the pulsation of the right ventricle, and believed it to be an exaggeration of the normal reflux taking place at the tricuspid orifice, as held by Hunter and others. This valuable contribution was entirely overlooked until brought to light by Walshe some years afterwards.

As bearing upon some of the aspects of our subject, it may be here mentioned that Louis, in 1829, published an elaborate treatise ("Recherches Anatomiques, &c., sur la Gastro-entérite," tom. i.) in which he pointed out the softened condition of the cardiac muscle in certain fevers. The softening varied greatly in degree up to a point at which the heart was universally affected, was quite flaccid, and became like a wet cloth (*linge mouillé*). In the minor degrees only the left ventricle seemed affected, but, even in the more marked cases, the left still maintained its degenerated supremacy. He found this condition of heart to a greater or less extent in various acute diseases.

The next material advance in cardiac knowledge was made by Hope ("A Treatise on Diseases of the Heart," &c., 1st ed., 1831), and it was from his time, and largely by his work, that accurate knowledge of the diseases of the heart became more generally diffused and valued throughout the profession. His experiments demonstrating the cause of the second sound, and the mode of production of regurgitant murmurs, must ever remain as part of the most valuable and admirable work done in this department of medicine. It is not, however, my purpose here to pass in review Hope's work as a whole, but to confine myself to that part of it bearing upon the inquiry in hand.

He was the first to recognise that a regurgitant murmur might result from a dilated orifice "consequent on dilatation of the ventricle rendering the valve, otherwise healthy,

incapable of closing it" (4th ed., p. 77), although he considered it a very rare condition. Tricuspid regurgitation might thus, he held, be due to patency of the valves from ventricular dilatation; but, at the same time, he pointed out the extreme rarity of tricuspid murmur, as the valves are so seldom affected by organic change (p. 388). Thus, while recognising a possibility, it occupied but a small place in the practical bearings of the subject.

Laennec had noted the bellows sound in hypertrophy and dilatation. Hope held that the murmur in these cases was a systolic aortic one, due in part to the anæmia which often accompanied cardiac disease of long duration, but also in part to the changed form of the ventricle; "for, as the cavity is more spherical than natural, and its artery consequently rises more abruptly with respect to its internal surface, the currents of blood reflected from its sides meet in the orifice at more obtuse angles, and thus, by their collision, not only give rise to the murmur, but impede each other's passage into the vessel. For the latter reason, the pulse is sometimes small and weak, when the impulse of the heart is violent,—a paradox which has much perplexed authors" (p. 94). This is a repetition of one of Laennec's errors to which reference has already been made, and which will be considered more fully in the sequel.

He also found the bellows murmur in certain persons of a delicate, hysterical, hypochondriacal, or other temperament; and he pointed out that the one common feature present in these cases was anæmia. The anæmia in animals, consequent on their being experimentally bled, was associated with the murmur. The loss of blood entailed impoverishment and "attenuation" of that fluid, unfilled arteries, and accelerated cardiac action. Apply-

ing, as Hope always did, the scientific method of inductive reasoning, he thus came to regard these three concomitants of anæmia as the *cause* of the bellows murmur in the classes referred to. When the murmur was cardiac it occurred at the gateway of the arterial system, and with the first sound (p. 106). The continuous murmur which was described by Laennec, and which got the appellation of *bruit de diable* from Bouillaud, was also, as Hope pointed out, associated with anæmia. Dr. Ogier Ward of Birmingham, in 1837 (*Medical Gazette*, vol. xx.) was the first to ascribe this murmur to the veins. This was accepted and taught by Hope. Hope further showed how the murmur was affected in degree, might even be obliterated by properly applied pressure, and that the pressure could be exercised by putting the sterno-mastoid muscle on the stretch, or even by stretching the skin over the vein. But he seems to have been satisfied with this record of accurate observations without inquiring further into its mode of production; unless, indeed, it be assumed that the observations are to be taken in conjunction with the propositions given above as to the cause of the arterial and cardiac murmur.

He summarised his position thus: "Modified movement of the blood, attended with increased friction and vibration, is, in all circumstances, the cause of inorganic murmurs and tremors, whether in the heart, the arteries, or the veins, and whether ordinary, continuous, humming, or whistling" (p. 383). He placed the impulse of the right ventricle under the inferior part of the sternum, and that of the left between the fifth and seventh ribs (p. 273); while the tricuspid murmur was loudest on or near the sternum, on the same level as the mitral murmur (p. 388).

Dr. C. J. B. Williams ("Diseases of the Chest," London,

1st ed., 1828, 4th ed., 1840) contributed some valuable observations towards the elucidation of the physics of murmurs. In chlorotic and anæmic persons he considered the watery condition of the blood one factor in the ease with which murmurs were produced, for the more watery a fluid, the more readily was it thrown into sonorous vibrations by slight pressure (4th ed., p. 219). He disputed Hope's explanation of the cause of murmur in dilated hypertrophy of the heart. He considered tricuspid murmur to be very rarely present (p. 277).

Bouillaud ("Traité Clinique des Maladies du Cœur," Bruxelles, 1836) entirely repudiated Laennec's explanation, that the *bruit de soufflet* was due to spasm of the heart and vessels. In addition to finding this bruit accompanying narrowing at the various cardiac orifices, he found it associated with anæmia, chlorosis, &c. (p. 63), and also fully recognised that it was at times due to dilatation of the auriculo-ventricular openings, and consequent insufficiency of the valves (p. 346 *et seq.*). He considered the murmur as merely due to increase of friction, but he further made the interesting statement that, in the anæmic, chlorotic, and nervous cases, while there was present the condition of active friction, yet he was prepared to regard some at least of these murmurs as probably to be explained by the presence of valve insufficiency (pp. 64-65). The *bruit de diable* and the *bruit de soufflet intermittent*, heard in the neck, were thought by him to have their seat in the arteries, and after enumerating all the anatomical and other possibilities affecting their production, he pathetically concludes by saying, "there probably is present, what it has not been given to us to penetrate, and of which we abandon the research to observers more able and better versed than we are in the

various matters which belong to the special department of acoustics" (p. 78).

Skoda ("A Treatise on Auscultation and Percussion," 1st ed., 1839; 4th ed., Markham's trans., London, 1853) pointed out that the impulse of the right ventricle might be felt to the left of the sternum (p. 171); in fact, he explicitly stated that "the part of the thorax where the apex beats is over the left ventricle, while to the right of this is the right ventricle" (p. 184). This is a step towards accuracy, although even here the "apex-beat" is presumed to occupy the situation of the left ventricle; and this, as shall be seen further on, is not necessarily the case.

He held that the murmurs not due to organic changes "undoubtedly depend for the most part upon friction between the blood and the walls of the heart. But how this friction produces the murmurs has yet to be explained. A contraction of the heart more forcible or rapid than ordinary cannot of itself produce a murmur; and, on the contrary, murmurs may exist when the movements of the heart are slow. The opinion that murmurs are caused by a particular condition of the blood must be looked upon as hypothetical until it has been shown in what that particular condition of the blood consists. It is not true that a watery state of the blood is a cause of murmurs in the heart," because in patients with watery blood he did not always find it (p. 211).

Inorganic murmurs were present in chlorosis in particular, but he also noted them in the cachexia of cancer, in acute rheumatism, puerperal diseases, the onset of typhus, of small-pox, and other inflammatory diseases (211 *et seq.*). The arterial murmur, he thought, was due either to friction of the blood against the wall of the vessel or to vibrations of their walls from distension; at the same

time he noted that the murmur was specially heard when the muscles of the neck were on the stretch (p. 215). He accepted the venous origin of the *bruit de diable*, and says it is loudest "in the erect position and during inspiration, but is never heard when the jugular veins are distended with blood" (p. 221). The majority of murmurs in the pulmonary artery was held not to depend upon structural change in the vessel, but upon other causes hitherto unexplained (p. 247).

Latham ("Collected Works," vol. i., New Sydenham Society, 1876, 1st ed., 1845) made no advance in the subject of our present inquiry. To him an impoverished state of the blood produced endocardial, arterial and venous murmur; and he based his belief on the appearance of these murmurs after venesection.

He did not think it possible to differentiate between mitral and tricuspid murmurs by any difference in the direction in which they were respectively conducted (p. 27); in fact, he regarded diagnosis of the right heart as less reliable than that of the left. It must be remembered, however, that this position of Latham, and of some others, was the result of the belief so firmly held by them, that bellows or endocardial murmur was evidence of endocardial inflammation.

He seems, however, to have been the first to clearly describe a murmur, systolic in time, which was confined to the second rib and second space for one inch from the left edge of the sternum. The murmur was found by him in persons suspected of a phthisical tendency. And he asks whether it is produced in the pulmonary artery from disease of the vessel or from extraneous pressure (p. 40)?

Hughes, in a paper on "Anæmic Murmurs and their Diagnosis" (*Guy's Hospital Reports*, vol. vii., 1851), was

the first to doubt the generally received opinion, that the hæmic murmur had its seat at the aortic orifice. With reference to the area of its audition, he says "it is inaudible not only below the nipple and in the axilla, as are also some aortic sounds dependent upon organic causes; but, generally, at any rate, it is also inaudible, as organic aortic murmurs are not, in the ascending portion and the arch of the aorta" (p. 160-161). He asks "whether the pulmonary artery might not possibly be the vessel from which it originated," and thinks the acoustic difficulties presented by the theory of their aortic origin would be removed by placing the murmur at the pulmonic orifice. After briefly reviewing some of the current opinions regarding the cause of the anæmic murmur, he grants deterioration in the quality of the blood as a necessary element, and believes that the other essential element is increased velocity of current, and consequent increased "attrition;" for, he says, "the amount of fluid, and the area through which it passes, being equal, the passage cannot be effected quickly without producing more attrition than if it passed slowly; any more than a flock of sheep can be driven quickly through a narrow gateway without knocking more rudely against the gateposts, than if they passed slowly, and at their leisure" (pp. 166-167). The venous murmur he did not attempt to explain, but he recognised the fact that an arterial murmur could be produced in the carotid artery by the pressure of the stethoscope.

Stokes ("Diseases of the Heart," &c., Dublin, 1854), held it to be impossible to distinguish the tricuspid from the mitral murmur by any auscultatory evidence, but considered jugular pulsation as sufficient proof of tricuspid regurgitation, and that it was to be regarded simply as

an exaggeration of the normal reflux occurring at this orifice; for he too accepted the doctrine at that time in vogue, that the safety-valve action of the tricuspid was exercised in health (p. 202 *et seq.*).

Reluctantly he believed in "simple, uncomplicated dilatation;" but, among the causes of "complicated dilatation," he included a debilitated condition of the cardiac muscle (p. 276); and he clearly accepted the doctrine that dilatation might be a cause of valvular incompetence from enlargement of the orifice (p. 258).

He verified the observations made by Louis on the state of the heart in fevers; but, in addition, studied the auscultatory phenomena of these affections during their onset, progress, and recovery. He followed with the stethoscope the gradual weakening and disappearance of the first sound over the left ventricle at the apex, while it continued audible over the sternum, until finally it disappeared even here. He ascribed the disappearance of the sounds to softening of the cardiac muscle, while Graves ("Clinical Lectures," Dublin, 1864, reprinted from 1848 ed.) maintained that the phenomenon was due to debility of the heart, and not to softening (p. 192). He further noted the reappearance of the sounds in a reversed order (p. 378). To the practical physician this was one of the most important and far-reaching advances made in clinical medicine, and we cannot but sympathise, and in part agree, with the belief, which these observations engendered, namely, that it is more important to determine the mechanical and vital power of the heart than to specify accurately the seat and nature of a valve lesion (p. 232). This is, however, only a partial truth, for it is often impossible to estimate accurately the former without a knowledge of the latter.

Another interesting observation made by Stokes, and bearing on our subject, was the description by him of a bellows murmur present in the relapse of a certain fever. The murmur was heard mainly between the sternum and the apex and masked the first sound, while in other cases it was most loud at the base, and frequently became inaudible when the patient sat up. When the murmur was not present there was frequently a prolongation or modification of the systole, accompanied in some cases by a somewhat vermicular character of impulse. It was regarded as attributable to a depraved state of the blood, or as dependent on a dynamic condition of the heart (p. 421 *et seq.*). He was not quite satisfied with the opinion of Hope and Walshe as to the site of inorganic murmur, and he considered there were difficulties in the way of determining that a murmur was purely basic; and further, he advocated caution in regarding a mitral murmur as necessarily organic (p. 496 *et seq.*). Although his position is thus in many respects so accurate, he resorted to a theory of "want of concert" between heart and arteries to explain the want of proportion between impulse and pulse in certain functional derangements of the heart (pp. 514-515).

Among the French physicians, Beau ("Traité Experimental, &c., d'Auscultation," Paris, 1856) maintained with great energy, and contended at great length, for the idea which dominates all his work, namely, that murmur was due to increase of friction from a want of proportion between the volume of the blood wave and the capacity of the channel containing it. While he valued the researches of Andral and Gavarret on the diminution of the corpuscular richness of the blood as a cause of vascular murmur, he held that the murmur did not depend on this

state of blood, but on a condition of the arterial system accompanying the diminution of the blood corpuscles (p. 399). He repudiated *in toto* the conclusions of Ogier Ward and Hope, that the vascular murmur had its seat in the veins, and contended that its real seat was the arteries. He did not believe that it was possible to determine, by the site of the audition of a murmur, whether it belonged to the right or the left cavities (p. 305). The murmur present in dilatation of the heart was due, he held, to a relative narrowing at the ventriculo-arterial orifice, or, in other words, to an altered proportion between the cavity of the ventricle and the lumen of the artery, whereby the more voluminous blood wave propelled by the former caused an exaggerated friction at the orifice of and in the vessel (p. 309 *et seq.*).

On this same hypothesis of *surabondance* of the blood wave, he explained the vascular murmurs present after bleeding. His explanation of how this large blood wave is produced is of special interest in our present inquiry. He noted in his experiments that, if an animal was speedily bled to death, the heart was found firmly contracted and empty; if, however, the animal had been reduced by frequent bleedings at considerable intervals, the heart was found both dilated and hypertrophied, and its cavities contained blood. In the human subject he not only found *post-mortem* a similar dilatation and hypertrophy, but recognised the evidences of it during life. He ascribed the condition to atony and relaxation of the muscular tissue due to impoverished blood; but held that it was compensated for by the accompanying hypertrophy. Thus the increase in the capacity of the ventricle increased the volume of its contents, which,

being propelled by the hypertrophous ventricle, gave the increased blood wave necessary to his theory.

In chlorosis he held that the pathological state of the blood was comparable to the *polyémie séreuse* following upon hæmorrhage; and that it involved a like dilatation of the heart. An analogous *organopathique* state of the heart was held to account for the murmur present in hysteria, hypochondria, and in such febrile conditions as typhoid and other fevers (p. 440 *et seq.*).

Bellingham ("Diseases of the Heart," Dublin, 1857), is particularly clear and explicit in some of his definitions of the causes of inorganic *bruit de soufflet.* In the review of Corrigan's work he strikes, as it seems to me, the keynote of the inquiry, when he says that the only physical agent invariably in operation is increased friction between the blood and the parts through which or along which it passes (p. 141). At the orifice of the aorta diminished viscidity and increased rapidity generated sufficient friction for the production of murmur. Similar conditions produced in the large arteries a murmur analogous to that produced in one under ordinary circumstances by pressure (p. 150). His definition of the causes producing venous murmur is still better. In order to develop sufficient friction between the blood and the interior of a vein to produce a murmur, the structures covering the vein must be rendered tense—these structures being skin, platysma, and cervical fascia. This is effected by turning the head towards the opposite side and elevating the chin. By this proceeding the vein was put in a condition favourable by its tension to the ready production of murmur within by slight pressure. This pressure was provided by the pressure of the stethoscope, and the murmur varied in proportion to the pressure; and if the fascia, &c., were

relaxed, and the head bent forward, there was no murmur. The diminution or cessation of the continuous murmur in the recumbent posture was in part due to the lessening of the rapidity of the circulation accompanying this posture, but also to the relaxation of the fascia associated with it (p. 157 *et seq.*). We may take exception to some of this teaching, but it is the clearest and most rational, in the main, with which we are acquainted. He held murmur at the tricuspid orifice to be exceedingly rare from any cause whatever, and ascribed its rarity to the weakness of the right ventricle (pp. 143-144).

Flint ("A Practical Treatise on the Diagnosis, &c., of Diseases of the Heart," Philadelphia, 1859) regarded epigastric pulsation, and pulsation under the lower part of the sternum, as indicative of enlargement of the right ventricle (p. 55 *et seq.*). Venous pulsation was often diagnostic of the degree in which the right chambers were affected in consequence of disease of the left heart, and he held that it was, as a rule, due to tricuspid regurgitation, although at times it might be due, as Hope held, to a transmitted impulse caused by the recoil of the valves upon the blood in a full auricle (p. 142 *et seq.*).

Inorganic murmur, he believed, was due to "an abnormal change in the composition and properties of the blood. The precise nature of the change is perhaps not positively ascertained." He was sceptical of the existence of dynamic murmurs; but, if they did exist, they were produced at the mitral orifice. The cardiac inorganic murmur he located in the pulmonary artery or aorta, or both combined (p. 220 *et seq.*).

Sibson's investigations ("Reynolds' System of Medicine," vol. iv.) into what may be termed the medical anatomy of the heart have not been equalled by any worker in this

country, and are so accurate and reliable that they can hardly be superseded. We have from him the most accurate definition of the areas with which we are here specially concerned. To quote his own words—"the tricuspid murmur is usually present over the body of the heart, or, in other terms, over the right ventricle; and extends from the lower half of the sternum to a line a little within the nipple, which line corresponds with the ventricular septum, and from the third to the sixth cartilage" (p. 463). This murmur sometimes extended to the right of the sternum. He differentiated the "immediate or tricuspid murmur" from the "transmitted or mitral murmur," by the latter being masked by the first sound of the right ventricle, while the former is inseparably incorporated with the first sound of the right ventricle, and begins with an accent (p. 467). The murmur in the second left space which, as has been seen, was first described by Latham, and subsequently held by Hughes to be produced in the pulmonary artery in some anæmics, was known to Sibson, and he referred it without reserve to the pulmonary artery, and ascribed it, when appearing later in the sequence of cardiac phenomena, to diminution of the whole volume of the blood and of the red corpuscles. When murmurs appeared early in the aorta or pulmonary artery he attributed it, in the former vessel, to lessened power of the ventricle, and lessened contents of the artery; while, in the latter, it was due to "reasons yet to be ascertained." He further noted the presence of a pulmonary murmur when the tricuspid murmur was disappearing, and considered it then as indicating lessened tension of the artery.

Parrot (*Archives Générales de Médecine*, Août, 1866) was the first to advance the opinion that the cardiac mur-

mur in anæmia was a tricuspid regurgitant one. He placed its site of greatest loudness in the fourth left space, but it sometimes attained its maximum in the fifth or third space. The murmur was propagated upwards and towards the right, in the direction of the right sterno-clavicular articulation. It was not heard to the left of the nipple. In support of this he referred to the readiness with which the tricuspid valves become incompetent, as shown by jugular pulsation; and contended that the situation of the murmur was over the tricuspid orifice, and that it was propagated in the direction of the great veins. The murmur heard in certain nervous and febrile conditions was also held by him to be tricuspid in origin.

With reference to the manner in which tricuspid incompetence was produced, he held that it was consequent on enlargement of the right ventricle, and therefore of the auriculo-ventricular orifice, thus leading to inability of the valve to close it; and that this enlargement of the ventricle was due to a relaxation of the cardiac muscle brought about by the impoverishment of the blood, and he quotes authorities to confirm his opinion.

He further held that this right-sided regurgitation led to diminished and insufficient supply of blood to the left heart, the ulterior consequence of which was that a lessened quantity was launched into the arteries, and that this lowered the arterial tension and gave the feeble pulse so frequent in anæmia. In fevers he did not believe the murmur to be a result of anæmia—at least, at the outset—but attributed it to a vaso-motor influence dilating the vessels, the consequence of which was engorgement of the right cavities, and the production in this way of a tricuspid regurgitant murmur.

In an unduly lofty tribute to the left ventricle, about which we shall have something further to say, he said: "it always rises to the height of its work. The left ventricle does not surrender; it does not fail the heart; it is the other cavities which fail it" (p. 153).

The work done by Walshe is so important, and the editions of his work ("Diseases of the Heart," &c.) extend over such a prolonged period, that it is impossible to give his work its proper chronological place without occupying more space than can be devoted to it here.

In his earlier days he was one of the first to teach that the limits of the heart could be made out by deep percussion. In his hands this attained great accuracy, and supplied much more valuable information than the determination of the superficial dulness which, up to his time, had been the method pursued.

He pointed out that extreme hypertrophy of the right ventricle might produce an impulse so much to the left of the sternum as to simulate enlargement of the left side; but even this he regarded as exceptional (p. 31). He also taught that it was not an uncommon feature of weak, fatty, and dilated hearts to have an undulatory impulse, or a "pseudo-undulation," over the precordia (p. 23). He, however, looked for evidence of an enlarged right side to epigastric pulsation and dulness to the right of the sternum (p. 327 *et seq.*); while jugular pulsation and a weakened pulmonary second sound were regarded as evidence of tricuspid incompetence; but the murmur denoting this condition was rarely met with, and when present, was heard over the lower part of the sternum, and was nearly, if not completely, inaudible at the left apex (p. 101). The first sound at the apex might be entirely masked by a systolic murmur, while at the right apex (above the

xiphoid cartilage) the first sound might be quite distinct (p. 85).

Clinically, he divided inorganic intra-cardiac murmurs into hæmic and dynamic.

The hæmic murmur was systolic in time, and was heard at the base of the heart in the aortic and pulmonary areas (second right and second left spaces), sometimes as low as the apex, but never, in his experience, beyond this point, and the murmurs were relegated to the orifices of the aorta and pulmonary artery (pp. 92-93). This murmur he found in many various conditions such as chlorosis, starvation, hæmorrhage, continued fever, the exanthemata, pneumonia, and acute rheumatism. He did not offer any lucid explanation of them, but confined himself mainly to a statement of the conditions under which they were present. In fact, he leaves their mode of production to be inferred from his consideration of the causes of the venous murmur.

The second or dynamic class of intra-cardiac murmur included those murmurs which could not be explained on any assumption of impoverished blood, and yet could not be classed under the title of "organic," always, of course, understanding that this term signified endocardial change appreciable on dissection. It included the apex murmur heard in cases of chorea; in cases of dilated and hypertrophous left ventricle; in others of flabby and possibly fatty heart; and in certain hysterical females and some males. Here it is necessary to observe that he considered mitral insufficiency and murmur, due to enlargement of the orifice, as of very rare occurrence (p. 99), so that this class must be distinctly excluded from the dynamic series.

In his section on the venous system he ascribes jugular distension to tricuspid regurgitation and dilatation of the

right cavities, the latter being in his opinion "a more rare" cause (p. 136). He disagreed with Hope's view that the venous pulse was transmitted through the auricle by the recoil of the tricuspid valve upon its contained blood.

The influence of posture on the venous murmur, was very clearly brought out by him. Any posture which stretched the vein intensified the murmur; while it could be modified or entirely suspended by properly applied pressure (p. 141). The conditions which Hope formulated as being necessary to the production of the venous murmur were not accepted by Walshe as final, and although compelled reluctantly to concede that the condition of the blood must have an important influence, he yet believed "the real mechanism of these murmurs to be obscure enough" (p. 147).

Monneret ("Traité de Pathologie Générale," 1857), quoted by Parrot, placed the murmur due to diminution of red corpuscles at the origin of the aorta.

Gendrin ("Leçons sur les Maladies du Cœur," 1841), quoted by Parrot, attributed the murmurs to changes in the composition of the blood.

Potain ("Dictionnaire Encyclopédique des Sciences Médicales," tom. iv.), also quoted by Parrot, placed the murmur at the arterial orifices, especially at the aortic orifice.

Von Dusch ("Lehrbuch der Herzkrankheiten," Leipzig, 1868), believed that the valves might be incompetent from dilatation of the orifices, and included chlorosis and febrile diseases amongst the causes producing dilatation (p. 120 *et seq.*). He placed the tricuspid murmur over the sternum, at the level of the fourth rib.

Among the later writers on cardiac disease in this country, we shall only consider the following:—

Hayden ("Diseases of the Heart and of the Aorta," Dublin, 1875), considered tricuspid murmurs as not infrequent, and located them over the left half of the lower third of the sternum, the adjoining fifth and sixth cartilages for an inch, in the space between them, and slightly in the fourth space (pp. 238-239). The pulmonic murmur he placed over the second and third ribs and the intermediate space, and regarded it as anæmic. He did not admit the doctrine held by so many of those who preceded him, that simple dilatation of the ventricle was enough of itself, without either narrowing or roughness, to create murmur at the auriculo-ventricular orifices (p. 173).

Non-organic murmurs he divided into hæmic and dynamic, as Walshe had done. The former were due to alteration in the quantity or quality of the blood, and might be cardiac, arterial, or venous (p. 245). The cardiac hæmic murmurs he held to be "invariably basic, loudest at mid-sternum or in the anatomical site of the orifices of the aorta and pulmonary artery," and that they had a diffusion area of three or four inches in diameter. All basic murmurs not due to disorganisation of the aortic orifice, he classed as hæmic (p. 275). The hæmic murmurs produced in the arteries and veins were regarded by him as the combined result of increased friction in the blood stream and increased vibration of the vessels (p. 265), but he acknowledged that it is vain to try to explain why these causes operate with effect exclusively in anæmic and spanæmic states (p. 265).

Dynamic murmurs were exclusively cardiac, and situated at the auriculo-ventricular orifices, and invariably regurgitant. The doctrine held by Stokes, Walshe, and others that the apex murmur present in chorea was due to an

affection of the muscular apparatus of the heart, either of the musculli papillares or of the walls, whereby either the one or the other contracted irregularly, was disputed by Hayden, who maintained that the murmur was due to "atony, or partial yielding of the walls of the left ventricle at the acme of systole" (p. 275). Its immediate causes were anæmia, debility, and muscular atony, and a similar murmur might be associated with purpura, masturbation, excessive tobacco-smoking, and other depressing causes (p. 281). The deranged mechanism which permitted this murmur he defined thus—"A yielding of a particular portion of the walls of the ventricle during the centripetal movement which takes place in the act of contraction, may so alter the direction in which one or both of the musculli papillares act upon the segments of the mitral valve, as totally to invert their function, by rendering them effective agents, not in closing but in opening the orifice of communication with the auricle. Such yielding may be due either to a want of sufficient contractile power in a particular part of the walls of the ventricle, or to ataxy or want of co-ordination in the contraction of the different portions of those walls" (p. 282). This murmur is placed by him at the apex; but Nixon, with whose observations he almost entirely agreed, seems to have placed it over the body of the ventricle, and both agreed that the murmur generally became inaudible when the patient sat up.

Among the causes of "primary" dilatation he includes anæmia and certain other debilitating circumstances; and the apex or mitral murmur which may be present in these cases, he explains on the same principle as those denominated dynamic, namely, by a "lifting" of one of the valve curtains by the musculli papillares attached to it (p. 558 *et seq.*).

Dr. Balfour ("Diseases of the Heart," London, 1876), holds that pulsation beneath the lower part of the sternum, with disappearance of the apex beat, reveals dilatation of the right ventricle, but that the impulse of the ventricle may also be communicated to the liver and visible in the right hypochondrium and epigastrium. He places the tricuspid auscultatory area over the lower part of the sternum, especially along its left edge (pp. 28 and 176 *et seq.*).

The first effect of a rheumatic attack on the cardiac muscle is, he holds, relaxation and consequent regurgitation from dilatation, with murmur in the mitral area, or "more often in the auricular area" (p. 148).

With reference to the endocardial murmurs of chlorosis, he regards the murmur in the second left space as the most frequent, and to explain it he adopts Naunyn's suggestion, namely, that the cause of an organic mitral regurgitant murmur being sometimes loudest in this neighbourhood, is owing to the murmur being carried into the left auricular appendix. He contends that the murmur is not audible over the pulmonary artery, but over the left auricular appendix; for the murmur attains its maximum intensity one inch and a-half or more to the left of the sternum, which, he says, coincides with the position of the appendix, as it "pops up from behind" the pulmonary artery (p. 160 *et seq.*). He therefore holds that the murmur is one of mitral regurgitation propagated into the appendix (p. 162). The next murmur to appear he holds to be a systolic mitral one; that, further, there is "sometimes a distinct tricuspid murmur, always more or less undulation in the jugular veins, and, lastly, a systolic aortic murmur propagated into the carotid arteries" (2nd ed., p. 174).

He refers to the "primary and typical hæmic murmur" (2nd ed., p. 172), as if former workers, when considering the question of hæmic murmurs heard over the precordia, had confined their descriptions to a murmur localised in the second left space. This is not correct, and the observations made and the views held by former eminent workers on the subject cannot be tested by this standard.

Niemeyer ("A Text-Book of Practical Medicine," 8th ed. English trans., 1878), includes protracted chlorosis among the causes of cardiac dilatation (vol. i. p. 319); and the murmurs in dilatation he regarded as depending upon "the irregularity of the vibrations, into which the ill-stretched valves are thrown by the current of the blood" (p. 324). The cardiac murmurs in chlorosis he considered as most probably arising from "an abnormal tension of the valves and arterial walls;" and not to the altered condition of the blood (p. 745). Relative insufficiency of the tricuspid valves he considers to be rare, if it ever occur. *Real pulsation* of the jugular veins, however, he regarded as pathognostic of insufficiency; and murmur, when present, was heard over the lower part of the sternum (p. 360).

Dr. Byrom Bramwell, whose work on Diseases of the Heart was published some time after the greater part of this volume was in manuscript, discusses very fully and judicially both Dr. Balfour's and my views as to the site and the mode of production of the murmur present in the second left space in anæmia. He concludes that "the *sudden* propulsion of a *large* blood wave of *abnormal* (spanæmic) composition into the vessel (the pulmonary artery), which is probably, in some cases at least, dilated, seems an efficient cause for its production" (p. 189). Dr. Bramwell's position is thus, as regards the cause of

anæmic murmur, midway between that held by Hope and Beau respectively, and the discussion of their views in the following chapters will be found equally applicable to his. He, like Dr. Balfour, finds, in advanced cases, a murmur at both the auriculo-ventricular and at both the arterial orifices.

CHAPTER II.

THE CONDITION OF THE HEART IN DEBILITY, AND ITS RELATIONS TO THE THORACIC WALL.

IN the debility which accompanies febrile states, special opportunities are afforded of following the onset and course of the changes which take place in the circulation. The changes are so similar in anæmia and chlorosis to what they are in these conditions, that they may be considered collectively.

To begin, it seems but a truism to assert that one of the first circulatory signs of debility is a weakened pulse; and yet, it may be asked, to what extent is this realised, and in what degree are the precise changes implied in this proposition appreciated? It is here maintained that the radial pulse is the measure of the strength of the left ventricle, due care being taken that neither an abnormal arterial distribution nor an abnormally small vessel misleads the observer; and on this point it is important to bear in mind that slight abnormalities are much more common than is generally recognised. Auscultation is also an important means of estimating the power of the left ventricle, if proper points be selected for examination. Laennec valued

so highly the information obtained by auscultation, that that derived from the pulse was almost wholly discredited by him; in fact, the examination of the pulse as a means of estimating the condition of the heart was practically discarded by him. He auscultated the heart between the left costal cartilages, and, as he often found its action and sounds pronounced in that position when the pulse was small and weak, he fell into the error referred to. Even Hope made a like mistake, and explained the anomaly of strong impulse with weak pulse, in dilatation with hypertrophy, by the angularity of the currents at the mouth of the aorta impeding the blood stream. Adams pointed out the true explanation of this disproportion between impulse and pulse by referring the former to the right ventricle. Notwithstanding this, the mistake is still frequently committed of regarding the cardiac power as normal when the first sound over the body of the organ is distinct, even although associated with a pulse considerably below the healthy standard.

Laennec noted the *obliquity* of the cardiac sounds in certain fevers. It was Stokes, however, who accurately followed the sequence of the auscultatory signs in fever, and taught that the first sound became gradually weakened and lost at the apex, while it continued to be heard over the body of the heart, and that, at last, it was lost even here; and that the sound returned in the various areas in a reversed sequence. The importance of these observations cannot be over-estimated. I have frequently tested and verified the accuracy of them. The explanation of the phenomena is simple. The first sound at the normal site of the apex is, under ordinary conditions, the measure of the power of the left ventricle, and the conclusions drawn from auscultation agree with those

drawn from the pulse. As debility progresses the power of the ventricle diminishes, and along with it the first sound lessens in loudness. As a consequence of this the second sound appears intensified; and this, although mainly only a relative effect, is also, in part, due to an absolute increase in the intensity of the pulmonary second sound.

The lessening of the first sound is often best followed by noting that, at the various examinations, the area narrows over which the sound is propagated outwards. The narrowing of the area of audition is due, as has been said, to the advancing debility of the ventricle, but also, in part, to the ventricle acquiring a deeper place in the chest consequent on the changes which have taken place in the relation of the various parts of the heart to the thoracic wall, as shall be seen later.

When the first sound is lost at the apex, the only guide to the state of the ventricle, apart from the evidence of the absence of the sound, is the pulse, and it is an absolutely safe and reliable one. But the first sound, after it is lost at the apex may continue distinct and fairly strong over the body of the heart,—that is, between the normal site of the apex and the left or right edge of the sternum. This area corresponds, according to Sibson, and according to my own observations, with the body of the right ventricle, and the first sound heard here is undoubtedly produced by its contraction, and it cannot be taken as a measure of the strength of the left ventricle. This explains the error which Laennec made, and which has been repeated by so many since his day.

The so-called "apex-beat," which has obtained the unfortunate importance of being regarded as a land-mark, is frequently a most unreliable guide as to the position of

the true apex of the heart; and in all states of debility it is seldom to be trusted.

The best method to follow, when there is reason to believe that a precordial pulsation is not apical, is to define the outer limit of deep dulness in the one or two spaces inferior to the nipple, then to auscultate just within the outer limit of this dull area, and to draw from the observations made there our deductions as to the power of the ventricle. It is unnecessary to do more than state here that the deep dulness reveals accurately the limit of the left edge of the heart, and that that line is formed by the left ventricle as a rule. If what is called the "apex-beat" falls about an inch or more within this line, it is not the impulse of the left ventricle, but of the right, which makes itself visible. This is the method which I have practised for years, and have found it of the utmost value in estimating the power of the left ventricle in typhoid and other fevers, and, in fact, in all conditions where the state of the heart forms an important element in treatment and in prognosis.

The returning power of a debilitated heart forms one of the most interesting of auscultatory studies, and often affords a more definite indication of the daily rate of recovery than any other means of observation at our disposal. The first sound reappears and gradually regains power, first over the body of the heart to the left of the sternum, then the area of audition extends more or less gradually to the apex, and finally extends outwards towards the axilla.

In estimating by auscultation the power of the heart, allowance has to be made for the variation in the relations of the heart to the thoracic wall in different individuals. In some persons the heart occupies such a superficial

position, and is so partially covered by lung, that not only can the modifications of its sounds be followed with the utmost ease, but the right ventricle reveals its changes by more or less extended pulsation. On the other hand, in persons with deep chests, the organ is placed deeply, and is so covered by lung, that its various phases are much less easily followed by ordinary physical examination; but even in these much can be learned either directly or by inference.

The constancy with which the heart participates in, and indeed is the leading factor in the production of subjective phenomena in debility, from whatever cause it may originate, seems to be but imperfectly realised, and does not obtain that careful consideration which, apart from its intrinsic interest, is demanded by it, seeing the organ holds the foremost place in what has been happily termed the "tripod of life." In the debility accompanying even brief pyrexia the organ presents very manifest evidence of its sympathetic response to even slight disturbance of the normal health. The suddenness with which this occurs and the like suddenness with which it disappears on the subsidence of the condition producing it, show very strikingly the readiness with which the heart is affected by conditions which may in themselves be regarded as practically unimportant.

The more precise nature of these changes may now be more fully considered.

In the normal condition there is no precordial pulsation except that localised within a narrow area at the apex. There is also neither episternal pulsation, nor pulsation above the right clavicle. In the abnormal conditions at present under consideration, the first sign of abnormality is the appearance of pulsation in one or other or both of

the two last-mentioned positions. This pulsation occurs along the course of the large veins, and is synchronous with the cardiac systole. That it is produced in the veins is beyond question, for the physical qualities of the large arteries make it impossible for them to be affected to an appreciable extent by the slight disturbance in which this pulsation may be noted. But further, if additional proof be required of the veins being the site of this pulsation, in a more advanced stage, and at each cardiac systole, the course of the external jugular becomes visible as a thin blue line which disappears between each systole. If such observations have been made on a person in the upright or sitting posture, it will be found that, on placing him in the recumbent posture, the external jugular remains visible during the entire cardiac cycle, while still presenting the systolic pulsation. In addition, the episternal and the supraclavicular areas of pulsation are distinctly fuller. The annexed tracing was taken by me from such a pulsation above the

clavicle. It is evident that it presents no resemblance to an arterial one, but on the contrary, is essentially similar to those given by Friedreich (*Deutsches Archiv für klinische Medicin*, I. Band, 1866) as specimens of venous tracings. According to this author, and to Gibson (Transactions Medico-Chirurgical Society of Edinburgh, 1882), the first wave "*a*" in the tracing is due to auricular systole; while the wave "*b*" to "*c*," with the broad summit, is due to ven-

tricular systole. I would add to this, that the character of the wave "*b*" to "*c*" is due to the reflux from the ventricle having to traverse the auricle in such a manner as to produce a wave resembling that obtained from the radial artery in certain cases of aortic aneurism (*vide* Marey's *Circulation du Sang*, with which my own experience coincides).

This excessive fulness of the venous trunks implies that the right auricle is in a similar state; which further implies that the efficient outlet for its contents into the ventricle, which normally exists, is interfered with. This means, no matter what view be taken of the action of the auricle, that it is not to a normal extent relieved of its contained blood during the ventricular diastole, and the only explanation which it is possible to give of this is, that there is no room in the ventricle for it. Yet it cannot be assumed that the cavity of the ventricle is diminished in capacity, so we are forced to conclude that it contains a quantity of blood above what it normally does at the termination of systole.

This increase in the volume of the residual blood in the ventricle is the result of several factors:—*first*, heightened tension in the pulmonary circuit, from causes hereafter considered, and inability on the part of the right ventricle to do more than partially cope with it; *second*, enlargement of its cavity, from relaxation of its muscular wall by its participation in the systemic condition producing the general debility; *third*, an increased rapidity of action consequent on direct or indirect nervous or other influences not allowing the enlarged cavity sufficient time to empty itself. These causes may be regarded as mutually dependent, and acting and reacting on each other. The important point to recognise and to grasp the significance of, is

the increase in the residual blood, and the enlargement of the ventricle.

Clinical evidence of the foregoing is afforded by persons with thin-walled chests.

After the appearance of the pulsations referred to as visible at the root of the neck, and indeed sometimes appearing simultaneously with them or antecedently to them, pulsation appears to the left of the sternum, in the second, third, fourth and perhaps fifth spaces, or in any of them, more commonly, although not invariably, in the upper than in the lower of these, should it be absent from any. The pulsation extends the farther to the left the lower the interspace, and reaches sometimes to the nipple line.

In the second space the pulsation is in part due to the pulmonary artery, and in part to the upper portion of the conus arteriosus of the right ventricle; but, when present early in the case, may be due entirely to the former. In the third, fourth and fifth spaces, the pulsation is produced by the body of the right ventricle.

It was doubtless this pulsation which led Laennec to under-estimate the information to be obtained from the pulse; and it is still a common error to estimate the cardiac strength by inspection, palpation and auscultation in this region.

In the perusal of cardiac literature, one is struck with the almost unvarying repetition of the earliest statements as to the indications to be sought for of enlargement of the right ventricle. The routine formula is: pulsation at the lower part of the sternum, and perhaps in the epigastrium. Here and there throughout the literature on the subject there is a recognition of the fact that the right heart may be seen beating to the left of the sternum; but

it was very falteringly, if at all, applied; and in practice the ancient formula was used.

Sibson's work, on what may be called the medical anatomy of the heart, is the most elaborate and careful work in the English language, and it is surprising that work of such a kind, and so accurate, should not have permeated and modelled the study of cardiac conditions more thoroughly than it seems to have done.

Epigastric pulsation, and pulsation at the lower part of the sternum, are very positive evidences of right-sided enlargement, but of a high degree of enlargement, which, in a still higher degree, is marked by pulsation in the region of the liver. A less degree of dilatation is, however, marked by pulsation in the spaces already enumerated. Proof of this last statement will be submitted later.

Pulsation in the second left space was long ago pointed out by Blakiston as sometimes probably due to the left auricular appendix. Naunyn has anew advanced this to explain the pulsation in this space in lesions at the mitral orifice. In England, Dr. G. W. Balfour has adopted this view, and has applied it to explain the pulsation in this space in debility and anæmia. Dr. Balfour has even gone further, and speaks of the pulsation above the fourth rib to the left of the sternum as auricular. Above the fourth rib is the anatomical position given by Walshe to the left auricle ("Diseases of the Heart," 3rd edit., p. 4); and he says: "Direct physical evidence of dilatation of either auricle is only to be had by percussion in the natural sites of those cavities" (p. 328); and he mentions one case where pulsation in the third left space, close to the sternum, was regarded as auricular, as it was not synchronous with the ventricular systole (p. 34). Hayden says: "From the anatomical position of the left auricle, dilatation of the

chamber must be difficult of recognition by independent signs; but, in conjunction with those of mitral narrowing, the existence of continuous feeble undulatory pulsation in the second and third intercostal spaces of the left side, about one inch from the sternum, may be regarded as evidence of this condition" ("Diseases of the Heart," &c., p. 576). Stokes says, with reference to the left auricle: "I have already remarked on the difficulty which the left auricle offers in any attempt to discover the enlargement by physical signs. This condition can only be inferred," &c. ("Diseases of the Heart," p. 273 *et seq.*). Schrötter says: "The left auricle is so hidden out of reach, that when it is dilated there are no signs which can be recognised by percussion" ("Ziemssen's Cyclopædia of Medicine," vol. vi. p. 213). Flint, while he does not absolutely discard all idea of auricular impulse, thinks the pulsation in the second left space much more reasonably referable to the ventricles or the pulmonary artery, as the auricle is deeply placed behind the large vessels ("A Practical Treatise on the Diagnosis, &c., of Diseases of the Heart," 1859, p. 53). Von Dusch (p. 5) also considers the auricle as not accessible during life. Dr. G. A. Gibson says: "The left auricle cannot in the same way give evidence of its condition; and, moreover, being deeply buried behind the ventricles and great vessels, changes of size are almost beyond the possibility of detection" (*Edinburgh Medical Journal*, August, 1882, p. 127).

It is thus seen that authors are divided, but that the weight of authority is against Walshe, Hayden, and Balfour. It is to be regretted that Dr. Balfour did not study the position of the heart after death, for, had he done so, he would certainly not have followed Walshe's teaching, and he would have found it unnecessary to

seek in the depths of the thorax for the cause of "pulsation and percussion dulness" above the fourth rib. The belief that the left auricle pulsates against the chest wall is so palpably inaccurate, that it would be superfluous to set one's-self seriously to refute it.

The propositions which I submit here are, that the right ventricle occupies the interspace above the fourth rib for some distance from the left edge of the sternum; that the pulmonary artery shares the second space with it, or that the artery may be carried higher, leaving the space to be occupied by the ventricle alone. And further, that, in the conditions under consideration, the left auricular appendix, instead of being nearer the chest wall, and sometimes pulsating against it, as Naunyn and Balfour have maintained, is further removed from it. The following *post-mortem* observations are submitted in proof of this:—

Observation I.—Mr. A., æt. 65, died of general paralysis. The second left space was occupied by the conus arteriosus and the pulmonary artery for one inch and a-half from the sternal edge, the junction of the two running diagonally across the space from above outwards and downwards. The left auricular appendix was invisible. The second right space was occupied by the appendix of the right auricle, which also lay under the sternum opposite this space. The aorta did not become uncovered by the appendix until it (the aorta) reached the level of the second rib. In the third right space the outer wall of the right auricle was fully one inch and a-quarter from the edge of the sternum. The apex of the heart was opposite the fourth space and fifth rib. By drawing the left wall of the pericardium outwards, a view of the heart in partial profile was obtained, when part of the left appendix, about the size of the tip of the little finger, was seen nestling

against the left postero-lateral aspect of the pulmonary artery; a line drawn from the second rib, where the cartilage had been severed from it, traversed a course of two inches before reaching this point. Both auricles contained fluid blood. All the chambers of the heart were relaxed and somewhat dilated. The valves were healthy. The muscular fibres showed various degrees of fatty degeneration when examined microscopically.

Observation II.—J. F., æt. 35, male, died of exhaustion consequent to "surgical kidneys." The body was plump, and there was considerable subcutaneous fat, which, as well as the skin, had the peculiar yellow hue and waxy appearance often noted in pernicious anæmia. The second left space was occupied for about one inch and a-half by the conus arteriosus and pulmonary artery, the junction of the two traversing the space. The left appendix was invisible; but its tip could be seen by obtaining a profile view, as in the preceding case. The second right space was occupied by the appendix of the right auricle, which entirely covered the aorta here. The right auricle extended for about one inch outwards in the third right space.

Observation III.—G. N., æt. 46, a male, died of pernicious anæmia. The conus arteriosus occupied the second left space, and the origin of the pulmonary artery was under the second rib. The left appendix was invisible, and its tip was only revealed by drawing aside the conus arteriosus and adjoining artery. The heart was very fatty, with a mottled endocardium. Its cavities were relaxed and dilated. It weighed 12½ oz.

Observation IV.—J. S., æt. 35, female, died of pernicious anæmia. The conus arteriosus occupied the second left space for about one inch and a-half, and extended upwards

under the second rib, under which rib also lay the origin of the pulmonary artery. The left appendix was invisible, and could only be seen by turning the heart round and removing about an ounce of fluid which the pericardium contained. The heart was fatty and dilated.

Observation V.—A man, aged 30, died of pernicious anæmia. The origin of the pulmonary artery was under the second rib. The conus arteriosus occupied the second left space for fully two inches. The left appendix was not visible, and the heart had to be turned upwards to bring it into view.

Observation VI.—A boy aged 7 years and 9 months died of purpura hæmorrhagica. The origin of the pulmonary artery was under the second rib, while the adjoining part of the right ventricle extended some distance outwards in the second space. The appendix of the auricle was visible, but was not unduly prominent, and was not enlarged.

It is well to mention here that the left appendix is relatively much more prominent in young persons than in adults.

The following observation is of much interest as proving absolutely that the conus arteriosus occupies, in the conditions under consideration, the second left space during life—

Observation VII.—Joseph S., æt. 72, died of cardiac syncope. The part of the conus arteriosus adjoining the pulmonary artery was in the second left space, and was fixed in this situation by old standing pericardial adhesions. There were no adhesions elsewhere. The origin of the pulmonary artery was under the second rib. The heart was opened *in situ*, when all the chambers were found to be full of fluid blood and soft black clot. The

organ was much enlarged, and all its chambers were dilated, while its muscular substance was markedly fatty, and the wall of the left ventricle was much diminished in thickness at the apex.

The foregoing cases are but a small portion of those which have come under my notice, but they are typical.

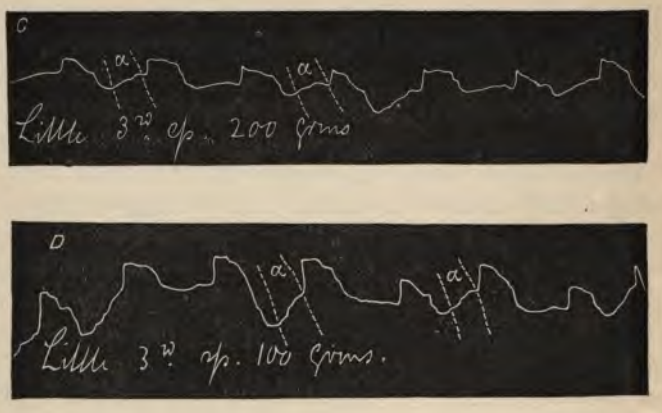
There can indeed be no doubt whatever that the relations I have indicated are those found in the conditions specified. That being the case, it is a grave and important clinical error, dependent upon erroneous conceptions as to the behaviour of the heart under morbid conditions, to regard a pulsation to the left of the sternum in the second and third spaces as being produced by the auricle. Further, although it is granted that the tip of the left appendix can, in normal conditions, be transfixed by passing a needle into the second left space some distance to the left of the sternum, in the conditions referred to it is far removed from the chest wall, and cannot possibly pulsate against it. Even in normal conditions it is at least exceedingly doubtful whether the left appendix approaches so close to the chest wall as some assume it does, but this point I do not propose to enter upon here. Whatever may be the normal position and normal proximity to the thoracic wall, in all states of debility, where increased tension and abnormal distension of the pulmonary artery are early events, as will be seen, the increase in the diameter of the vessel instead of lessening increases the space which the appendix has to travel to reach the chest wall; for, it will be remembered, that the appendix comes forward from behind the pulmonary artery, and has to traverse a space at least equal to the diameter of that vessel. And all extension, no matter how slight, of the right ventricle to the left, similarly affects the relative

position of the appendix, and further aids in overlapping it. For the proper comprehension of this change, it is necessary to understand that the posterior attachments of the heart, formed by the pulmonary veins, make the left auricle a fixed point, so that the heart at its root is not readily moved.

The assertion made by Dr. Balfour that the auricular appendix is enlarged to any considerable degree has yet to be proved; for there are no recorded observations either on this, or on the other interesting point, as to which diameter is most increased. For the acceptance of the doctrine of auricular appendix pulsation it would be necessary to prove that the appendix is elongated, as the most earnest believer in the theory can scarcely claim that more than the tip of the structure reaches the chest. I have made no precise measurements; but, after examining a large number of hearts in all morbid conditions, I am satisfied that when the appendix is enlarged it is, as a rule, more so in its transverse than in its longitudinal diameter; in fact, it has often seemed to me that the enlargement of the auricle was in part effected by drawing upon the appendix, and that the tendency was more towards obliteration of this normal diverticulum than towards enlargement of it.

Clinical proof of the accuracy of my contentions as to the parts of the heart producing pulsation in the second and third left spaces can be obtained by tracings. On this point it is, however, necessary to warn observers that, in the majority of cases, the tracings obtained are of no value whatever; but in patients with thin chest walls, wide spaces, and a heart not deeply placed, tracings can be obtained fully corroborating the facts obtained by *post-mortem* observations.

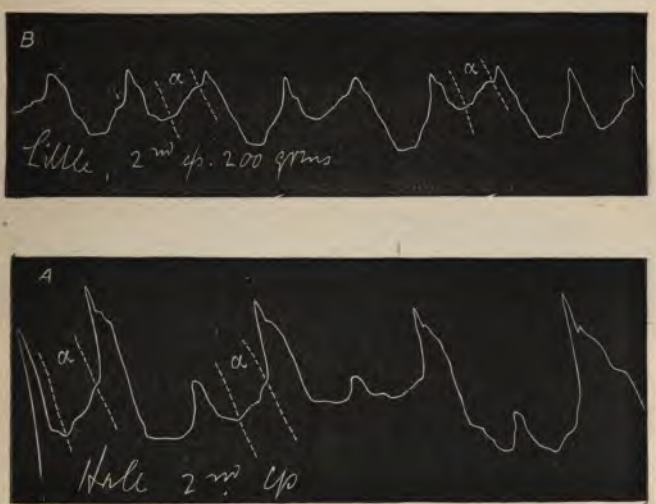
Tracings C and D were taken from Case III., hereafter referred to (p. 58), with different degrees of pressure, from the third left space some distance to the left of the sternum where pulsation was visible. The space "*a*" between the dotted lines, and which immediately precedes the systolic shock, represents the latter part of the time occupied by the filling of the ventricle, and includes the auricular systole. The systolic shock is represented by the perpendicular line immediately following, while the



prolonged and blunt summit represents the duration of ventricular systole, the tracing owing its completeness in this respect to the heart being well in contact with the parietes. This tracing bears a complete resemblance to those taken by Marey from the right ventricle (*Circulation du Sang*).

Tracing B was taken from the second left space of the same patient. Tracing A was taken from the second space of Case VI., hereafter referred to (p. 59). In

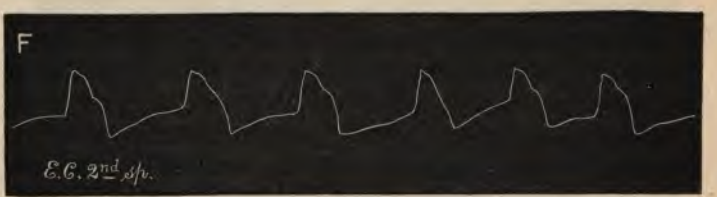
both the space *a* is well marked, and is to be explained in the same way as the similar part in C and D, so it appears evident that at that moment the button of the sphygmograph was over the right ventricle. B and A have however a sharp summit, instead of the long, blunt top of C and D; it is therefore inferred that the ventricle was no longer under the instrument, but had retreated



from its position, which is exactly what would be expected; for it is an acknowledged fact that the conus arteriosus, during its contraction, descends about an interspace, carrying the pulmonary artery with it. The pulmonary artery is the only part of the heart which could come under the instrument when the contraction of the conus arteriosus takes place; and the arterial character of the down-line, with its tidal and dicrotic waves, is to be

attributed, in part at least, to this fact. It is not sought to exaggerate the significance of this, for although this part of the tracing receives its character from the impulse communicated by the closure of the semilunar valves, there are other conditions in which a similar down-line may be obtained, and at considerable distance from the pulmonary artery, and which is to be explained by the transmission of the shock of the closure of the semilunar valves to the part on which the instrument rests.

Tracing F was taken one inch from the sternal edge in the second left space in Case X., referred to hereafter



(p. 63), and from the varying degree of the bluntness of the summit, would point to the conclusion that the ventricle was contracting with unequal power; the more acute the summit, the stronger the systole, and the more complete the downward movement of the conus arteriosus. This view was further strengthened both by digital examination and by auscultation. The tracing, as a whole, much more resembles a ventricular tracing than the two preceding tracings taken from this space, and my belief at the time was that the ventricle occupied the space more completely than in my former cases.

Having thus satisfied ourselves as to the condition and relations to the chest wall of the right side of the heart

it is now necessary to turn our attention to the left side. In this study we cannot obtain our results by the method of direct observation, which guided our preceding investigation, but we hope to attain as high a degree of precision by legitimate inference and accurate deduction.

In debility, the guide which the clinician depends upon above all others is the pulse. The feeble pulse implies languid mental functions, readily fatigued muscular functions, impaired digestive functions, and so forth. But, as has been already indicated, the state of the pulse depends not so much on any inherent quality in itself, as on the condition of the organ which regulates its rapidity, and on the volume of its blood-wave. A slow heart gives a slow pulse; a strong heart gives a voluminous pulse-wave; and the converse must be equally true. This requires no demonstration, it is accepted as an axiom as soon as it is formulated. But what is the condition of the organ at the centre of the circulation which accompanies the weak pulse?

In the first place, it is assumed that the cavity of the left ventricle has not diminished in capacity, and therefore holds as much blood as it does in robust health. In robust health we know that the ventricle empties itself of its contained blood so completely, that its propulsion into the arterial system produces a wave of considerable volume and energy, and that the pulse-wave is the necessary result of such a volume of blood passing into the aorta. On the other hand, if the pulse-wave be small and weak, there is a smaller volume of blood propelled into the aorta with the cardiac systole, and the unavoidable inference is, that the ventricle does not empty itself as it does in ordinary and healthy conditions, so that we thus have, as we had on the right side, an abnormal volume of residual blood at the termination of each systole.

Increased rapidity often accompanies, but it does not compensate for, diminished power. The increase of rapidity prevents what might be a dangerous degree of stasis in the ventricles, but it does not help in any way to the more complete emptying of them.

In the normal condition, the whole ventricular capacity is required for the blood ready to be poured into it from the auricle during ventricular diastole. In the abnormal conditions under consideration, the auricle cannot to the same degree relieve itself, owing to the increase of the residual blood in the ventricle already occupying part of the space ordinarily provided for the blood coming from the auricle. That is to say, if the residual blood in the ventricle is increased by a given amount, the residual blood in the auricle becomes increased by a like amount. The consequence is, that the auricle is not capable of receiving the amount of blood which normally pours into it synchronously with each systole of the right ventricle (Foster's "Human Physiology"). The result is, that the tension in the pulmonary circuit is increased, and the pulmonary artery abnormally distended. At this stage, were it not that the right ventricle readily adapted itself, or yielded to the opposition in front of it, the result would necessarily and speedily be disastrous, by rupture of pulmonary capillaries. And, as a rule, it is not until the tension in the pulmonary circuit has been increased, that the right ventricle yields. We do not endorse Parrot's position that it is the right ventricle which fails the heart, but, on the contrary, hold that it adapts itself to the failure of the left ventricle.

Having thus seen that the residual blood in the chambers on the left side of the heart must be increased, we may now pass to the consideration of the condition of the muscle and of the cavities.

At the extreme limit on one side may be placed the cases on which Louis' admirable work was based, namely, the softened and disorganised heart of putrid fevers. Shading off from these were fatal cases of allied or similar disease, in which he always found the heart more or less softened, the condition being always more marked on the left than on the right side, while it might be exclusively confined to the left. He also found a degree of this degeneration present in various acute diseases.

As has been already mentioned, Stokes traced the auscultatory phenomena in fever, and explained them as produced by degrees of softening less severe than in the fatal form, while Graves maintained that the phenomena were due to debility alone. Beau, in his experimental inquiries into the effects produced by bleeding, found the heart, after repeated bleedings extending over a considerable period, dilated and hypertrophied, and containing blood in its cavities after death. He found a like condition in the human subject, and explained it as due to atony and relaxation of the muscular tissue. It must be remembered that these results are very different from those observed in animals speedily bled to death. In chlorosis the condition was similar; the heart was dilated and hypertrophied, the former being due to atony from impoverished blood. Parrot says that "Bamberger, Friedreich, Vogel, and Stark think that in chlorosis the fibres of the ventricular walls undergo relaxation, brought about by disordered nutrition, from diminution of the coloured corpuscles in the blood" (*Archives Générales de Médecine*, Août, 1866, p. 153), and he endorses this. Schrötter says: "After certain febrile affections, there ensues, on account of the breaking down of the muscular fibres of the heart into a molecular detritus, and the consequent relaxation of the

muscular tissue, an acute dilatation of the heart. Doubtless the changes which occur in chlorosis may be explained in this manner" ("Ziemssen's Cyclopædia of the Practice of Medicine," vol. vi. p. 201). Von Dusch (*loc. cit.*, p. 121) likewise refers to the molecular changes in the cardiac muscle in chlorosis. Hayden places anæmia at the head of the list of causes producing "primary dilatation" (*loc. cit.*, p. 558 *et seq.*). Dr. Balfour says that in chlorosis there is "relaxation of the whole muscular system, including the heart" (*loc. cit.*, 2nd ed., p. 173).

My own observations, both at the bedside and after death, point to a like conclusion. Some of them have already been detailed, others will be referred to later.

The clinical points which I have depended upon as indicating increase in the size of the heart are, increase in the area of cardiac dulness and extension of the area of normal pulsation, especially an outward or downward extension of it. After death the evidence relied upon has been the state of the cavities which are usually found relaxed and flaccid, and microscopical examination as to the state of the muscular fibres. In fatal cases of pernicious anæmia and analogous disease, the heart is found to be flaccid from relaxation of its muscle, and the fibres are found swollen and in a condition of granular or fatty degeneration.

In the normal state the involuntary muscle of internal organs, equally with voluntary muscle, is in a condition of tonicity. If from any cause this tonicity be interfered with, dilatation of the organ follows. This is well seen in the uterus after delivery, and is a recognised condition in such hollow organs as the stomach and the bladder. Similar effects must attend the atonicity of the cardiac muscle. This is not to be understood as a stretching but as a

relaxation, and the degree to which it is carried is in direct proportion to the degree of the cause producing it. Whenever the cardiac action is weak, not only is the propulsive power of the organ lowered, but its normal tonicity must likewise be lessened, and the result of this is assuredly dilatation of its cavities. The amount of this may be small at first, but the cause continuing, the effect tends to increase, not only owing to the continuance of the cause, but also owing to the residual blood acting as a further dilating agent.

Before leaving this part of the subject, it is necessary to consider a strange error which pervades the whole literature of this subject. All the authorities speak of the hypertrophy attending the dilatation of debility, and refer to it as a compensatory process. This is the view Beau took of it. Hope, Stokes, Walshe, and Hayden, took up much the same position. Parrot says: "The left ventricle always rises to the height of its work" (*loc. cit.*, p. 153). Dr. Balfour speaks of dilatation being first compensated for by the "reserve force" in the ventricle, and afterwards by hypertrophy (p. 173). I make bold to say that such a view, emanating from authorities of eminence, who devoted a considerable part of their great abilities to the elucidation of cardiac conditions, is a cause of legitimate amazement. In forming and contending for an opinion diametrically opposed to this, I am fully conscious of the apparent audacity of my position; but, on the other hand, I so appreciate the great work done by them, that I am assured of its enduring value, even should a section here and there of that work have to be corrected by a later knowledge.

It is necessary to draw a very marked and clear distinction between hypertrophy and enlargement of the heart

when our knowledge has to be applied clinically. Enlargement is increase in size and in weight, whereas hypertrophy is not only this, but, in addition, increase of functional power. The increase in size and weight of the heart, in the conditions under consideration, is granted without reserve, is, in fact, insisted on, for we have seen, in the *post-mortem* observations already given, that the organ answers to this description in pernicious anæmia, and in other debilitating diseases. But that there is increase of functional power we entirely dispute. How is it possible to conceive that the heart, which, as has been seen, yields with an almost startling rapidity to certain abnormal conditions implying debility, and the evidence of which is a weakened propulsive power and a muscular atonicity—how is it possible, we ask, for such a heart to manifest hypertrophy? True, the condition may induce, or be accompanied by an enlargement of the organ, as is the case in pernicious anæmia, but that enlargement, instead of signifying increased power, indicates, on the contrary, diminished power. It would be as correct to say that the plump arm of the chlorotic female is a muscular hypertrophy similar to the blacksmith's arm, as to apply the same term to the heart of the former as is applied to the heart of the latter.

And how does the view held by Dr. Balfour, of a reserve force in a debilitated heart show itself? Every clinician knows that in debility the slightest effort wearies, and that the increased pulse-rate consequent thereon is not an indication of a reserve force being called into action, but a manifestation of inability of the organ to meet the strain put upon it; and the subjective symptoms referable to the heart alone may compel the individual to desist from an effort which, if prolonged, would lead to an irrecover-

able degree of dilatation. In acute disease, the balance is so finely adjusted in the recumbent position that setting your patient up may induce an instantaneously fatal syncope—there is little evidence of a reserve energy here in the sense Dr. Balfour seeks to apply it. Our whole contention has been that the heart and pulse are the indicators of debility, and those who have believed in hypertrophy, took the pulse as the indication of debility without seeing the want of concord thus existing between their pathological notions and their clinical observations. A hypertrophied heart and a pulse denoting debility, combined in the same individual, is a palpable impossibility, and the belief in such a combination is one of the gravest of errors. I fully recognise that the hypertrophied heart of other conditions enters, at a certain stage in its history, on a degenerative phase, marking the onset of its downward career; but with this condition we have nothing to do here, save to point out that even there the point of moment to our patient is, that the degenerative process, manifested by the signs of debility already referred to, should be recognised and treated. The fact of the organ having been hypertrophied, and being enlarged, are factors taken into account in the etiological retrospect, and in the prognosis; but it is no longer the factor demanding the attention of the therapeutist. In debility from anæmia, chlorosis, &c., there is no compensatory hypertrophy. Debility may be, and often is, the more or less prolonged last phase of hypertrophy; but hypertrophy cannot be a phase of debility, although enlargement may be, and often is. Flint evidently had a glimmering of this, for he says: "It may be fairly doubted if the palpitation incident to anæmia be ever competent in itself to induce hypertrophy" (*loc. cit.*, p. 29).

APPENDIX.

THE following is a list of the fatal cases of Pernicious Anæmia which occurred in the Edinburgh Royal Infirmary between October, 1878, and April, 1884, in which a *post-mortem* examination of the body was made:—

Case Book 32.

No. of Case.	Age.	Weight of Heart.	Remarks.
84	Male, 45	15 ozs.	Muscle said not to be fatty.
27	„ 45	15½ ozs.	Muscle friable.

Case Book 31.

325	Female, 29	12½ ozs.	Muscle fatty.
298	Male, 34	16½ ozs.	Muscle fatty and soft.
121	„ 28	17 ozs.	Do. do.

Case Book 30.

317	Male, 60	14½ ozs.	Muscle fatty.
165	Female, 62	13 ozs.	Do. do.
163	„ 55	13½ ozs.	Do. do.
104	Male, 57	17 ozs.	Do. do.

Case Book 29.

267	Male, 17	6 ozs.	Muscle fatty.

(Patient's height, 4 ft. 7 in.)

From these it appears that the heart is increased in size and in weight to a very considerable degree, and that the muscle in all the cases was fatty.

In only one case, namely the first in the list, is the heart said not to be fatty. There is, however, considerable doubt as to the accuracy of this observation.

I am indebted to Dr. Byrom Bramwell for his kind permission to use the foregoing, and also for the trouble he very kindly took in going through the Registers with me.

They agree with my own observations, and bear out my contention, that the enlargement of the heart in these cases is not a true hypertrophy.

CHAPTER III.

THE SITE AND MECHANISM OF THE CARDIAC MURMURS IN DEBILITY AND ANÆMIA.

I PROPOSE in this chapter to consider the murmurs frequently present in debility from various causes, but which are most commonly known as occurring in anæmia and chlorosis. I shall confine myself mainly to the cardiac murmurs; and shall, in the first place, briefly review the leading points in the history of the subject.

Laennec attributed the bellows sound in the heart, when there was no organic lesion, to spasm of the orifices. This explanation, however, was soon rejected as untenable. Hope formulated the conditions associated with murmur consequent upon loss of blood as follows:—Unfilled vessels, velocity of current, and deterioration in the quality of the blood. This formula was regarded by himself, and became generally accepted, as the expression of the mechanism of the murmur, which was believed to have its seat at the origin of the aorta. With various modifications this has been tenderly handed down to the present time. All the ingenuity of physicians and others, for several decades, seems to have been brought to bear upon the elucidation

of the phenomena. Some upheld the all-powerfulness of velocity of current; others contended for rippling current and vibrating walls; others found increased friction at the arterial gateway and along its ramifications. All possible combinations of these, with the almost universal addition of deteriorated blood, were applied to the solution of the problem. Beau attempted to explain them, as well as all other murmurs, by a want of correspondence between the blood-wave and the channel into which it was propelled. Sibson believed in the lessened contents of the aorta, and lessened power of the ventricle as the cause of the murmur. While others, like Bouillaud, abandoned the attempt to explain them; or like Walshe, after making the attempt, conclude that the conditions are obscure enough. Skoda thought inorganic murmur was probably due to increase of friction between the blood and the vascular or cardiac wall, although he knew not how it was produced, and denied that the conditions formulated by Hope were sufficient. His scepticism, however, left undisturbed the current of opinion, and his views were only mentioned to be condemned.

Twenty years after the appearance of Hope's first edition, Hughes argued that the pulmonary artery might be the seat of the hæmic murmur, and so successfully did he do so that thenceforth the pulmonary artery was credited, with a share at least, in what to that time had been the sole and almost undisputed possession of the aorta.

In 1866 Parrot attacked the position, arguing from clinical observations that the murmur was a tricuspid regurgitant one. In England at least this view made no material effect on current opinion. Although Parrot's paper was a valuable contribution to our present subject, it contains errors of such a nature that they tend in a

considerable degree to detract from its merits, and to obstruct the acceptance of his teaching. The regurgitation which he found in anæmia, and other debilitated states, was attributed by him solely to failure of the right ventricle; and he held that this failure led to an imperfect blood supply to the left heart. He failed to grasp the fact, on which so much stress has been laid here, that the left heart shared in the primary debility; that the so-called hypertrophy did not compensate for that debility; and that, if in any way it was to be regarded as an effort at compensation, the effort was abortive and spent itself in increasing size, and not in augmenting power. Another extraordinary view he advanced was as to the means by which tricuspid regurgitation occurred in fevers, where he believed the murmur was due to regurgitation, and not to altered blood, as was then generally believed. His explanation was that, owing to the vaso-motor condition, the capillaries were released, and thus contained an excess of blood, which, finding its way to the right heart, led to its engorgement; and the right heart always failing, as he thought, to rise to any extra work thrown upon it, dilated and made the auriculo-ventricular valve incompetent. Such a view does not require serious refutation. It was an exaggerated development of the common belief that the right heart is so weak that it is readily rendered unequal to its work, and even unable to produce audible sound by its contraction.

In 1868 Naunyn (*Berl. Klin. Wochenschrift*, 1868) advanced the doctrine that in cases of mitral regurgitation, when the murmur was loudest above the fourth left rib, it was due to its propagation through the auricle into the appendix, and was heard in this situation from the proximity of the greatly dilated appendix to the chest

wall. This view of the phenomenon has been accepted by Rosenstein (Ziemssen's Cyclopædia of Medicine), Guttmann ("Handbook of Physical Diagnosis," Sydenham Society's translation), and others.

Dr. G. W. Balfour applied the foregoing to the explanation of the murmur present in the second left space in chlorosis and allied conditions, advocating that the murmur was a mitral regurgitant one carried into the appendix by the regurgitating stream; and, advanced in proof of this, that the point of maximum intensity of the murmur was an inch or more to the left of the sternum, and that this point was to the left of the pulmonary artery, and coincided with the anatomical position of the auricular appendix. He further maintained that the appendix could frequently be seen and felt pulsating in this position. He has not, however, in his hitherto published works given a single instance in which his contention is supported by *post-mortem* observations made with the heart *in situ*. I have in the preceding chapter advanced certain arguments against the probability of the appendix reaching the chest wall so as to pulsate there. I have submitted tracings of the pulsation in the second left space from over the seat of the murmur, and offered the only possible explanation of them. Further, I have shown that, in pernicious anæmia and other exhausting diseases, the auricular appendix instead of being so enlarged as to reach the chest wall was not enlarged, and was far removed from the chest wall; and this, I contend, is the common relation of parts when the heart is dilated and relaxed. I readily grant the theory to be a very beautiful one, and acknowledge that at one time I held by it; but a larger clinical experience, and a large number of *post-mortem* observa-

tions compelled me to abandon it as not in harmony with the facts of pathological anatomy.

Dr. Balfour holds, that the first endocardial murmur present in chlorosis is one of mitral regurgitation heard in the position referred to, and which is called by him auricular. The next murmur which appears he regards as mitral, and as heard in its own proper area. Further, there is sometimes a distinct tricuspid murmur; while at a later stage he places murmurs in the aorta and pulmonary artery, and believes they are then due to "large blood-waves" entering these vessels (*Brit. Med. Journal*, 1882, vol. ii. p. 353). Dr. Balfour thus gives us in advanced chlorosis a combination of all former opinions, and finds a murmur at all the cardiac orifices.

His "large blood-wave" theory is a revival of Beau's idea of *l'ondée surabondante*, and is the outcome of that grave error to which we have already referred, namely, that in debility, with relaxation of the cardiac muscle, there was a compensatory hypertrophy. Nothing could accentuate more markedly the false pathology of such views than the contention, that in the most advanced stages of anæmia, murmurs are produced at the arterial orifices by the magnitude of the blood-wave, when we know, both by the pulse and by auscultation, that the heart is incapable of the strong systole needed for the large blood-wave.

The inaccuracy or incompleteness of other opinions will be seen as the subject is developed in its more formative and positive aspects; and we propose to begin this investigation by briefly recording the auscultatory and other phenomena in a series of cases.

Case I. *Pulsation in the second left space: no murmur.* —A. H., aged 15, had been confined to bed for weeks, suffering from Bright's disease, with obstinate hæmaturia.

She was very pale, but plump. There was slight precordial tremor, becoming pulsation in the second left space, where it extended for over an inch from the sternal edge. There was marked accentuation of the pulmonary second sound, but no murmur. There was no venous pulsation, and no *bruit de diable.*

CASE II. *Pulsation in the second left space, and systolic impurity.* — J. R., æt. 18, showed signs of commencing phthisis at the right apex, and was subject to recurring and slight hæmoptysis, with rise of temperature. After one of these attacks there was precordial pulsation, visible when lying down, from the fifth to the second left space. In the second space the pulsation was distinctly divided into two parts; the inner extended for a quarter of an inch to the left of the sternum, and conveyed the idea of being continuous with the pulsation in the other spaces, while beyond this there was a feebler, and seemingly separate, pulsation extending for one inch. The external jugular vein was visible, but did not oscillate. The pulmonary second sound was loudly accentuated, and the first sound in the second left space (the pulmonary area) was prolonged and occasionally murmuring. In some days this passed off, along with the rise of temperature, only leaving a slightly accentuated pulmonary second sound.

CASE III. *Pulsation and murmur in the second left space, only present after exertion.*—Thomas L., aged 11, was suffering from a mild attack of articular rheumatism. On the third day of treatment the pulse was 80, and the temperature 99·2°. There was pulsation in the external jugulars and at the root of the neck. There was a slight and diffused heaving visible over the precordia. The apex could be felt in the fourth space, an inch outside the nipple. There was no murmur, but the pulmonary second

sound was loudly accentuated, and slapping in character. On slight exertion, either by rising up in bed and lying down again several times, or by taking several forced inspirations, the precordial heaving became more marked, and, especially during expiration, there was a marked pulsation in the second left space, most marked about an inch from the sternum, and over this a loud and localised systolic murmur. Tracing B, in the preceding chapter was taken from the site of pulsation and murmur.

CASE IV. *Bruit de diable: murmur in second left space, and no pulsation.*—Miss G. H., æt. 17, was anæmic, but plump; there was undulation at the root of the neck and for some distance along the course of the internal jugular vein. There was a *bruit de diable.* There was no precordial pulsation; but a rough systolic murmur was audible in the second left space, followed by a loudly accentuated pulmonary second sound. The murmur entirely vanished even on partial inspiration.

CASE V. *Murmur in the second left space: no pulsation: faint apex murmur.*—Miss F., æt. 23, was very pale and chlorotic. The pulse was 100 to 120 (excited), empty, and jerking. The external jugular vein pulsated synchronously with the radial pulse, and the vein collapsed and disappeared between each systole. There was a *bruit de diable.* There was no precordial pulsation. There was a faint and localised systolic murmur at the apex, and a murmur of the same rhythm in the second left space, completely lost on partial inspiration. The pulmonary second sound was accentuated. When she stood up there was no pulse visible in the external jugular veins, and no murmur was audible over the heart.

CASE VI. *Pulsation in second, third, and fourth left spaces: systolic murmur in second space.*—James H.,

aged 13, was suffering from a mild attack of typhoid fever. He had a thin-walled chest, with marked interspaces. The pulse was 80, regular, and of fair volume and force. There was precordial pulsation in the second, third, and fourth left spaces: in the second, it extended outwards for an inch and a-half; in the fourth, it reached one inch beyond the nipple. There was a systolic murmur confined to the second space, and loudest over the outer part of the pulsation. The pulmonary second sound was accentuated. There was oscillation in the external jugulars, but no venous murmur. The leading changes which occurred during the progress of the case were, that the apex pulsation descended to the fifth space, one inch outside the nipple line; that the murmur disappeared in a day or two for one day, and then reappeared; that it again vanished when the boy was at his worst, with a pulse of 60, very small and weak, and when the precordial pulsation was much less apparent. As he improved the murmur again appeared, and finally vanished when the rehabilitation of the heart was evidenced by a stronger and more frequent pulse, by the venous pulsation being reduced to a mere flicker, by the apex retreating under the fifth rib, and by the area of pulsation in the second left space lessening in extent. Tracing A was taken from the site of maximum pulsation and murmur in the second left space.

CASE VII. *Slight precordial pulsation: distension of veins: systolic murmur over body of heart, and in second and third right and second left spaces.*—Mrs. H., aged 33, was very pale and slightly chlorotic. Examined lying down, the external jugular vein was not only full, but so distended that it stood out, and did not collapse during any part of the circulatory cycle; it, of course, pul-

sated. There was faint precordial pulsation visible in the fourth left space inside the nipple, and in the third space one inch from the sternum. There was no apex-beat; but over its normal site there was a systolic murmur, lost one inch outside the nipple line; it was louder over the site of pulsation in the fourth space; less loud in the third and second spaces; also audible over the sternum from the level of the second space downwards; heard also in the second right and in the third right spaces, for nearly two inches from the sternal edge, and not present over the manubrium sterni nor in the carotids. It was louder in the second right than in the corresponding left space; and, in the third right space, it gradually lessened in intensity as the stethoscope was carried outwards, until it could only be detected as a very distant and faint blowing sound. The pulmonary and aortic second sounds were about equal in intensity, and neither of them were loud. There was a venous murmur between the origins of the sterno-mastoid muscle, presenting two distinct periods of increased loudness during a cardiac revolution. The veins running down in front of the trachea pulsated visibly. In the erect posture the external jugular was only marked by a thin blue line during ventricular systole, and no murmur could be heard anywhere over the precordia. The patient rapidly improved under appropriate treatment. The veins became much less engorged. The murmur disappeared first from the third and second right spaces and from over the sternum, until it was only present in the second, third and fourth left spaces.

CASE VIII. *Venous murmur: murmur over precordia and to right of sternum: murmur in carotid from pressure.*—Margaret H., aged 26, complained of the ordinary symptoms of debility and anæmia. She was

markedly chlorotic. When seen first on 18th February there was no precordial pulsation visible. There was pulsation in the external jugular vein and over the large veins at the root of the neck. There was a continuous venous murmur at the root of the neck. There was a systolic murmur at the apex which was conducted towards the axilla. The murmur was also present over the body of the heart to the left of the sternum as high as the second rib, also over the whole length of the sternum, being least loud over the manubrium. It was also present in the second, third and fourth right spaces, being loudest in the first of these, but propagated farther outwards in the third and fourth than in the second. A systolic murmur was produced in the carotid artery by the slightest pressure of the stethoscope. That the murmur was really thus produced was demonstrated by placing the finger over the vessel below the point on which the stethoscope rested, when it was found that the slightest digital pressure developed the murmur even when the stethoscope was resting so lightly as to make it otherwise inaudible. The tone of the murmur could be modified by the degree of pressure exerted by the finger. The murmurs were louder in the recumbent than in the sitting posture. On 9th March, the *bruit de diable* and venous distension had disappeared. There was a systolic murmur in the second left space when the patient lay down, and only during expiration. A faint systolic impurity was also audible over the middle of the sternum.

CASE IX. *Bruit de diable: murmur over precordia when recumbent: murmur in carotid by pressure.*—Miss B., aged 17, presented the ordinary symptoms and appearance of chlorosis. When in the erect posture there was a continuous venous murmur at the root of the neck,

but no murmur over the precordia. Lying down, there was a systolic murmur over the body of the heart; the jugulars were full and pulsating; and the venous murmur presented a distinct systolic accentuation. A murmur was readily produced in the carotid by pressure.

CASE X. *Pulsation second left space: venous distension: no murmur: pulmonary second felt.*—E. C., æt. 10, had a mild attack of articular rheumatism. There was pulsation in the second and third left spaces extending in the former for fully one inch from the sternum. There was also venous pulsation. Tracing E was taken from this case from the outer part of the pulsation in the second space; where also the clang of the accentuated second sound could be both heard and felt through the stethoscope, as if immediately beneath the instrument. Only once out of a number of observations was a faint systolic murmur heard in this space. The tracing from this case has been already referred to as resembling those taken from the ventricle, and we have no doubt it was taken from the conus arteriosus.

CASE XI. *Murmur over body of heart and in second left space.*—A man, aged 30, died of pernicious anæmia. The apex of the heart beat in the fifth interspace, half an inch outside the nipple line. Over the precordia to the left of the sternum there was a systolic murmur which was heard as high as the second space, in which space dulness extended outwards over an inch. For the *post-mortem* appearances see Observation V., p. 39.

CASE XII. *Showing in part the sequence of phenomena.*—A lad, aged 17, died of hæmorrhagic purpura. There was pulsation in the second, third and fourth left interspaces. Over the site of pulsation in the fourth space, the first sound had a slight impurity. At the apex the

first sound was faint and impure, the impurity being heard towards the axilla. There was a well-marked systolic murmur in the second left space, lost half an inch from the sternum, not audible on the adjoining part of the sternum or in the third space, and only heard during expiration. The pulmonary second sound was much accentuated; the veins in the neck were not distended. These notes were taken on the 7th or 8th of the month of admission. On the 12th, when the patient had become decidedly worse, there was no murmur or impurity audible at the apex or towards the axilla. There was, however, a systolic murmur in the second, third, and fourth left spaces which was markedly loudest in the first of these. The jugulars were now prominently pulsating. The murmur was also audible over part of the sternum and to the right of it. The precordial pulsation had also become more marked.

CASE XIII. *Showing in part the sequence of phenomena.*—A boy, aged 7 years and 9 months, died of hæmorrhagic purpura. On admission, the pulmonary second sound was accentuated, but there was no murmur over the cardiac region. Ten days after admission, when he had become much worse, there was a systolic murmur in the second, third and fourth left spaces, loudest in the second; it was also audible over part of the sternum, and was noted as present to the right of it.

Cases like the foregoing might be multiplied indefinitely, but these thirteen are sufficient to illustrate my position.

The sequence of phenomena revealed by the stethoscope is thus—first, accentuation of the pulmonary second sound, with perhaps a systolic impurity in the second left space; second, a systolic murmur in the second left space; third, murmur in the third and fourth left spaces in addition to

the second space; fourth, murmur from about the nipple line inwards over a triangular area, the left boundary of which is formed by a line drawn from below the nipple to a point in the second left space from one to two inches to the left of the sternum, and often heard at the same time over the sternum adjoining this area; fifth, murmur in the second right space ("aortic area"); sixth, murmur in the third and perhaps the fourth right space; seventh, murmur over the manubrium sterni; eighth, murmur beyond the apex towards the axilla.

In different cases the stages vary somewhat, and they frequently overlap each other.

The explanation of these various phenomena has now to be sought for.

I will say nothing on the subject of precordial pulsation, as not only its degree but its presence often depends largely on the physical qualities of the chest wall. When present it may, as has been already pointed out, give valuable information.

The accentuation of the pulmonary second sound is to be regarded as evidence that the tension in the pulmonary circuit has been increased. This is not, in the early stage, due to mitral regurgitation, but to accumulation of blood in the left heart, which, as has been seen, takes place as soon as the heart manifests its debility by a weakened systole. Accentuation is judged of in two ways: first, by comparison with the second sound in the second right space; second, by comparison with the first sound. Both aid in the formation of an opinion.

The murmur in the second left space is of great interest for many reasons. Hughes, as has been seen, was the first to claim for the pulmonary artery a share in the "basic murmur of chlorosis," and it has been granted to it by many

since his time; but the classic "basic murmur of chlorosis" had quite another position, as will be seen presently. The murmur to which I refer is a murmur which is heard in the second left space at a varying distance from the sternal edge, but seldom exceeding one inch and a-half. It is usually confined to this space, but may be heard over the rib above or below it, and is usually not heard over even the smallest bit of sternum adjoining the space. A common peculiarity of the murmur is, that, when the chest is elevated by inspiration, the murmur disappears, reappearing during expiration. Sometimes a partial and very shallow inspiration is accompanied by this disappearance; in other cases it only vanishes at the termination of a very deep inspiration; while again, in others, the deepest inspiration is not accompanied by its disappearance. The murmur is frequently not a perfectly smooth bellows sound, but is somewhat grating or harsh in character. Another interesting and important peculiarity is that it is very frequently inaudible when in the erect posture, but immediately becomes so in the recumbent posture. In fact, so much have I been impressed with this that I do not now regard any examination of the heart as conclusive or satisfactory unless the individual examined has been so in both positions. Some of my professional friends have been much struck with this after I had drawn their attention to the fact. The murmur is always followed by an accentuated pulmonary second sound. Pulsation may be present at the point where the murmur is audible, and then, by the finger or through the stethoscope, the clang of the second sound may be felt, conveying the impression that the act was occurring immediately beneath the finger or stethoscope. Comparison along the space, and above and below it, leaves no doubt as to the spot which coincides

with the origin of the vessel. Latham described this murmur. Sibson and others relegated it without question to the pulmonary artery. Quincke, quoted by Guttmann ("A Handbook of Physical Diagnosis," 1879, New Sydenham Society's translation), held that one of the causes of this pulmonic murmur was an abnormal proportion between the calibre of the conus arteriosus and the pulmonary artery. Dr. G. Balfour has referred the pulsation and the murmur to the left auricular appendix, but this theory has been already referred to at some length, and has been found to fail completely when brought to the test of *post-mortem* observation, and other methods of investigation. On the other hand, it has been shown that the pulmonary artery occupies the position which it is said not to occupy, and which is claimed for the appendix.

While we thus maintain that the pulmonary artery is the seat of this murmur, the question remains: How is it produced?

Sibson held that it was due to lessened blood in the vessel and lowered power of the right ventricle; but as he noted the appearance of the murmur when tricuspid reflux and murmur were disappearing, it is difficult to understand how he committed himself to an opinion so palpably erroneous. If reflux be disappearing, the ventricle must be recovering itself; so, instead of lessening, there is increasing power, and this cannot be associated with diminution but with maintenance of the quantity of blood in the pulmonary circuit.

Quincke and others have explained it by want of proportion between the conus arteriosus and the artery, which is the theory Beau universally applied, and, if true, should produce aortic murmur in all cases of dilatation of the left heart, and this is not found to be the case. In fact, were

this theory correct, we should expect at all times to find murmur at all the cardiac orifices in the direction of the normal blood current, as it passed from smaller into larger spaces or *vice versa*.

Dilatation of the pulmonary artery will not account for the murmur, for it is not found in persons suffering from emphysema, and who exhibit incontrovertible evidence of heightened tension in the pulmonary artery; nor indeed is it necessarily present in cases of organic disease of the left heart, with similar increase of tension.

Guttmann's theory, that it is due to "unequal tension of the sides of the artery" (*loc. cit.*, p. 287), is inconsistent with the most elementary physical laws.

The murmur further does not fit the formula enunciated by Hope as to the causation of "hæmic" murmurs, for there is not present the condition of unfilled vessel; and often there is not the other condition, namely, velocity of current; while its early appearance in pyrexial states excludes the other condition, deterioration in the quality of the blood to any considerable degree.

I may now consider what I believe to be the mode of production of the murmur. I have already contended that in debility there is an atonic dilatation of the cardiac cavities, and an increase in the residual blood in its various chambers. It will, therefore, be understood that, as the pulmonary artery arches over the left auricle, any increase in the size of the auricle, or any abnormal fulness of it, interferes with the normal play of the vessel. This is further understood when it is remembered that the pulmonary artery goes to a point at the root of the lungs, which, from its anatomical connections, is a fixed point. If anything therefore tends to increase the distance which the pulmonary artery has to travel, this point does not

change its position, but remains fixed; so that any change which may be necessary must take place by the movement of the origin of the vessel.

By injecting the heart *in situ* it is readily seen how fulness of the left auricle lifts the pulmonary artery towards the chest wall, and increases the space the vessel has to cover to reach the root of the lung. With the heart thus injected, and *in situ*, it is seen that the descent of the conus arteriosus, which normally carries the origin of the artery along with it for about an inch, cannot take place even to a small extent without interfering with the calibre of the vessel at its origin. This is in reality a flattening of the vessel by a greater or less approximation of its opposite surfaces in the same way as a piece of elastic tubing becomes flattened when stretched over anything placed behind it. It must not be forgotten that the tension in the right ventricle at the moment of systole is greater than the tension in the pulmonary artery (Foster's "Physiology"), so that the power of the ventricle producing this relatively greater tension is of course able to stretch, and in so doing to partially flatten the vessel.

There can, I think, be no doubt that an increase in the antero-posterior diameter of the left ventricle is a factor in the production of this effect by tending in a like manner to lift or push the pulmonary artery at its origin towards the chest wall.

The murmur thus becomes a measure of the degree of distension or dilatation of the left heart. The accentuation of the pulmonary second sound being an earlier indication of its condition.

The explanation here submitted provides the essential factor necessary to the production of murmur; and makes it dependent, not on the physical or chemical condition of

the blood, but on the degree in which the heart is yielding to the strain thrown upon it by any one of many various conditions. This cannot be regarded as a matter of small moment. It carries with it knowledge and appreciation of the dynamics of the organ upon which the circulation depends, and by which physical strength is measured. The condition may be induced by anæmic blood, but it accompanies many other conditions, conditions all presenting the common feature of debility, and it may even be the result of exhaustion from over-work or from strain. It takes away from the murmur the indefiniteness and inaccuracy of being regarded as "hæmic," and as due to ill-defined chemical changes, and gives it a place as a murmur dependent on mechanical changes, which may or may not have been induced by chemical alterations in the blood.

The influence of posture on the murmur is probably due to various factors. The recumbent posture aids the arterial circulation, as the same power gives a wave of greater amplitude along a horizontal than along a vertical plane. The venous circulation in the inferior vena cava is also aided by the removal of the gravity which acts powerfully on its contents. These two facts are clear. On the other hand, however, the influence which gravity exercises in aiding the blood-flow from auricle to ventricle, in the erect position, is removed in the recumbent one, so that the auricles are not relieved in the same way as the venous system of the inferior cava. That they are not is proved by what occurs on the right side when the individual is recumbent. The external jugular may then be seen to continue prominently distended during the entire cardiac cycle, showing conclusively that the auricle likewise continues full. From this we can legitimately infer that the left auricle is in a similar

condition. So that we thus have the conditions which I have contended are necessary for the production of the murmur.

The influence that inspiration often has in leading to the disappearance of the murmur is due to the elevation of the thorax, allowing the heart freer play. With the increase in the space between the heart and the chest wall, the lower segment of the heart is tilted more forwards than is possible when the space is contracted during expiration; the heart is thus able to get more in line with the pulmonary artery, and thus the *kink* which otherwise would occur is prevented.

The influence of the recumbent position in producing the murmur, and of inspiration in leading to its disappearance, absolutely negative any shred of remaining belief that the size of the pulmonary artery and the condition of the blood have any material influence on its production. At the same time, we grant the accuracy of Poiseuille's law that the molecular cohesion of the fluid is a factor in the ease with which certain murmurs may be produced; and especially is this to be granted in anæmia and chlorosis; but, without interference with calibre, murmur is not produced even in these cases.

After the pulmonary murmur has been present, perhaps for some time, a systolic murmur appears in the third and fourth left spaces in addition to the second. This region coincides with the position of the conus arteriosus and adjoining portion of the right ventricle. As the appearance of this murmur is associated with a lessening in the intensity of the pulmonary second sound, and by a diminution in the intensity of the pulmonary murmur, and often by a more marked pulsation in the veins of the neck, there can be no doubt that the murmur is one of

tricuspid regurgitation. It is heard here because this part of the thorax covers the part of the right ventricle which first reaches the chest wall when the ventricle is dilated. That this is the case can be seen by slowly injecting the exposed heart *in situ*.

This murmur may gradually extend until it is audible from the site of the normal apex to the sternum, and as high as the second left space or even rib; also over the sternum in its middle third or lower two-thirds. At the same time, it is audible to the right of the sternum, in the second right space (the so-called aortic area), and it then becomes audible, if not already so, in the third and fourth right spaces also.

Our *post-mortem* observations showed that the second right space was occupied by the appendix of the right auricle, which here covered the aorta. It thus appears that the murmur is not heard in the aorta, as is generally believed, but that it is heard over the right appendix, and is, in fact, a tricuspid murmur propagated into it. That the murmur is not aortic is further proved by its absence, in many cases, over the manubrium sterni and towards the left sterno-clavicular articulation, when a murmur of like intensity could not fail to be audible in these positions were it produced at the aortic orifice. A further proof of our contention is that the murmur appears in the third and perhaps fourth spaces, and that it gradually fades as the stethoscope is carried outwards until it is only audible as the faintest of breath sounds; and our *post-mortem* observations show that these spaces are occupied by the right auricle. We thus cannot avoid the conclusion that the murmur is a tricuspid regurgitant one carried into the auricle.

The murmur in the second left space, in the more

advanced stages, is heard over the conus arteriosus and not over the pulmonary artery. It is then tricuspid in origin. This change is brought about by the altered relations of the heart to the thoracic wall. As the right side dilates, the origin of the pulmonary artery is carried upwards and to the left; and the entire right heart undergoes a kind of rotatory movement from right to left, by which more and more of its surface is brought into contact with the chest wall. This process takes place from left to right until the substernal pulsation appears, which is acknowledged by all to be a sign of enlargement of the right heart, but which, we have already contended, only appears as a sign of great enlargement.

The fact of the pulmonary artery being carried upwards and outwards (to the left) is an additional proof of the accuracy of our contention as to the manner in which the primary murmur is produced in this interspace. Were the space the vessel has to traverse not increased by the distension of the parts behind it, this upward and outward displacement would crumple it transversely, and this only happens in a slight degree in the most advanced cases, and is probably due to the final dilatation preceding death. We have, however, seen the origin of the vessel carried so much forwards, upwards, and to the left as to make its course to the root of the lung a downward and backward one only.

It will be readily understood that this carrying of the origin of the vessel upwards and outwards relieves it from the dragging to which it was in the first place exposed, and that thereby the flattening of the vessel is prevented, and the murmur ceases to be produced in it.

This change of position occurs also in dilatation of the right heart from organic disease of the left, so that the

absence of the murmur in the pulmonary artery in these cases is not an argument of any value against my contention, as has been held by some.

When the murmur is audible over the manubrium sterni it is a sign of very great dilatation of the right heart. The murmur in this situation is probably never louder than it is lower down the sternum ; it is, in addition, not propagated into the carotids. These two facts negative the idea of its being of aortic origin.

We further hold that there is no carotid murmur in anæmia save that produced by the pressure of the stethoscope. This can be proved to be correct by very careful auscultation in the manner referred to in Cases VIII. and IX. We grant that the altered physical properties of the blood, and the diminution in the contents of the arteries, are factors in the ease with which arterial murmurs are produced by slight pressure. A beautiful illustration of the influence of the latter factor is afforded by such an artery as the femoral in aortic regurgitation, where the vessel is too far from the heart to have a murmur produced at the aortic orifice propagated to it, and yet where, with slight pressure, a double murmur is produced, the pressure for the production of the systolic murmur only requiring to be very slight, and much less than is required to produce a like murmur in a normally-filled vessel.

It will be thus seen that, by our contentions, the classic "basic murmur" of anæmia can no longer be regarded as an aortic murmur, but that it is in reality a tricuspid murmur.

When the systolic murmur is propagated outwards beyond the cardiac dulness, as it sometimes is, it is due to mitral regurgitation having become fully developed, for tricuspid murmur is not heard beyond the heart to the left.

That mitral regurgitation is present before mitral murmur is audible, as Dr. Balfour has maintained, is open to doubt. As dilatation increases, the efficiency of the mitral valve to close its orifice diminishes, either from enlargement of the orifice, or from the increased weakness of the ventricle leading to a diminution in the amount of contraction normally taking place at the orifice, and which is necessary to its complete occlusion by the valves. The result of this is that we have mitral murmur established.

I am inclined to think that Dr. Balfour has regarded the tricuspid as a mitral murmur. If its conduction outwards is to be taken, and this we hold to be the case, as the only reliable evidence of its presence at the mitral orifice, it is, as a rule, the last murmur to be developed, and is the least common and not the most common after the pulmonary murmur, as Dr. Balfour believes.

It is, therefore, submitted that the explanation here advanced of the murmurs in question is based on well-understood physical laws, and that the explanation is in harmony with the facts of pathological anatomy, meets the necessities of clinical observation, and gives the phenomena an interest and an importance which they did not possess so long as they were regarded as blood-murmurs due to ill-understood chemical changes in the circulating fluid. Further, our work does not run counter to the *observations* of former eminent workers in this field; on the contrary, their observations and definitions, and even difficulties, can be explained on the principles which have been detailed in the foregoing pages. We thus have the so-called cardiac hæmic murmurs reduced from complexity, confusion and obscurity, to order and simplicity.

Although the foregoing is held to be the explanation of

the cardiac murmurs which are present in many cases of debility and anæmia, it is perhaps desirable to state distinctly that there may be no murmurs audible over any part of the precordia. This absence of murmur may be due to an extreme degree of debility of the cardiac muscle. In other cases, it would seem to be attributable to the fact that the heart has not been subjected to continued strain. Many anæmics, in spite of languor and debility, continue to perform the daily round of duty and of toil, and when at last they seek advice it is found that the cardiac murmurs present their most typical features. In such cases there is the element of strain added to the impoverishment of the blood which nourishes the organ. The continued strain leads to an increasing dilatation of the organ, and thus it is that we frequently find both tricuspid and mitral regurgitation fully established. On the other hand, in anæmics who are so situated that they can submit to the sense of weariness and fatigue, the murmurs may not be nearly so well developed. In one of the cases recorded in this chapter, it was seen that in spite of prolonged hæmaturia and considerable anæmia there were no cardiac murmurs, because, as I believe, the patient was resting in bed, and there was thus no strain on the heart muscle. It is important to keep clearly in mind this element of strain, as it affords a valuable therapeutic indication, which, if appropriately met, gives usually much more speedy results than are obtained when it is ignored.

In febrile affections the presence of a murmur over the precordia, when the case is first seen, is often an indication that the patient's ordinary business has been persevered with in spite of malaise and indisposition. The murmur in these cases is the expression of the effect of elevated temperature and of strain combined. In cases of

a similar kind, which have been seen early, and where rest in bed has been insisted on, the febrile affection, whatever it be, may run its course without any murmur developing. Elevated temperature is present, but the other factor is wanting.

CHAPTER IV.

THE VASCULAR MURMURS IN CHLOROSIS AND ANÆMIA.

IN the preceding chapter it has been shown that the cardiac murmur, which alone is to be regarded as *truly* basic in position, and primary in point of time, is produced in the pulmonary artery.

The "basic murmur," as the term has been applied in cases of chlorosis, is not situated in the pulmonary artery, but has its seat at the tricuspid orifice.

The area of audition of the precordial murmur in many cases of chlorosis coincides, as will be seen in the following chapter, with that of tricuspid murmur due to incompetence from organic changes in the valves. As this is the case, and as there is other incontrovertible evidence of tricuspid regurgitation being present, it seems almost superfluous to contend more strongly that the murmur has its seat at the tricuspid orifice. The area over which the murmur is often heard coincides with the thoracic area which covers the right heart, except in those cases where a mitral regurgitant murmur is likewise present. In the latter case the murmur is propagated to the left beyond the cardiac area.

The loudness over the sternum of what is here called the tricuspid murmur, led to the opinion, first promulgated by Hope, that the murmur was basic in origin, and situated at the orifice of the aorta. Hughes, as has already been seen, tried to wrest from the aorta the exclusiveness of its possession, and to give the pulmonary artery a share therein; while Parrot tried to transfer its monopoly to the tricuspid orifice. They both partially succeeded in their efforts; but the belief in the existence of the aortic systolic murmur has with great constancy been adhered to down to the present day.

In some of the cases which have been detailed in the preceding chapter, the murmur heard over the precordia was either not audible, or only faintly so, over the manubrium sterni, while it was loud over the body of that bone. Cases illustrating this might be multiplied indefinitely, if necessary or desirable. If the observation be correct, and it is here maintained to be so, the murmur naturally falls into the category of tricuspid murmurs. It certainly cannot be allowed to retain its classic position at the aortic orifice. Were it situated there it would not only be as loud, but louder, over the manubrium than over the body of the sternum; and it is quite inconceivable that it should ever be inaudible over the former when present over the latter. The evidence presented by physical examination is, indeed, as a rule, very different to that presented by aortic systolic murmur.

The presence of a murmur in the second right space has always been taken as conclusive evidence that its origin was in the aorta. This is an error which has been handed on from authority to authority owing to the simple fact that in morbid conditions the altered relations of the various parts of the heart to the thoracic wall, and the

influence these alterations exercise in determining the areas over which murmurs are heard, have not been sufficiently studied. In part it was doubtless due to the idea, which pervades the earlier cardiac literature, that the right heart was so weak that its systole was not accompanied by sound, and that, consequently, tricuspid reflux was inaudible. Although there is not perhaps expressed a like poor estimate of the power of the right heart, there is still prevalent a very marked hesitation in diagnosing tricuspid murmurs, and murmur in the second right space continues to be regarded as necessarily aortic.

If the *post mortem* observations already detailed have been followed, it will be remembered, that in the second right space the aorta was under cover of the appendix or other part of the right auricle. The natural and ready explanation of the murmur in the second space is, therefore, that it is a tricuspid regurgitant one, carried by the regurgitating stream into the appendix of the auricle. In this case there is no doubt the appendix reaches the chest wall. The further proof of the accuracy of this contention is, that the murmur is also audible in the third and fourth right spaces, over an area the outer limit of which corresponds to the right wall of the auricle. Surely no more satisfactory or absolute proof could be afforded of our contention, and it appears to leave no room for doubt.

It was the wide area of audition of the murmur, coupled with the idea that it was aortic, or aortic and pulmonary combined, which led to the belief that the direction and the extent of the diffusion of the murmur were erratic in the extreme; and were either ruled by mysterious physical laws, or subject to no law. As soon as it is grasped that the first murmur heard over the precordia in anæmia and debility is, as a rule, produced in the pulmonary artery,

that the next murmur is a tricuspid regurgitant one, and that the area over which the latter is audible depends upon the degree of distension of the right heart, and the measure in which it approaches the thoracic wall, so soon do the precordial murmurs range themselves under simple and orderly law.

The varying degree in which the murmur is audible, even over the manubrium, depends entirely upon the degree of the distension of the right side of the heart.

There is, it is here contended, no aortic murmur in anæmia or chlorosis. The murmur which has been supposed to be aortic in origin is really tricuspid. What has been here shown to be the *primary* murmur is produced in the pulmonary artery, but it has not been, to the same extent, confounded with the tricuspid murmur as the so-called aortic murmur has been; neither was it this murmur which led to the belief that the *basic hæmic murmur* was aortic in origin. The classical basic murmur was undoubtedly what is here held to be the tricuspid murmur.

Having thus refuted the contention that the *hæmic* basic murmur is situated at the orifice of the aorta, it necessarily follows that the murmur in the carotid artery, which was supposed to be the aortic one conducted upwards, has likewise no existence. It is however not to be forgotten that, in several of the older descriptions of the *hæmic* or basic inorganic cardiac murmur, it was clearly brought out that the murmur was but feebly, if at all, conducted into the vessels of the neck. We may go further and say, that as it is not conducted into the carotid, it cannot have its origin at the aortic orifice; for that an aortic systolic murmur should be heard loudly over the sternum and not in the carotid is a physical impossibility. The arterial murmur was, however, frequently regarded as

being produced in the course of the vessels and as quite distinct from the cardiac basic murmur, and it requires great care in auscultating to determine that there is no murmur in the arteries. When the stethoscope is applied over the carotid a murmur is readily heard; but, if the pressure be lessened, the murmur will be found to disappear in proportion to the lightness with which the instrument presses, until, finally, it becomes quite inaudible. The impression then is, that the murmur has become inaudible from the instrument not being sufficiently in contact with the integuments. To demonstrate that such is not the case, it is necessary to apply the finger over the course of the vessel, below the point at which the stethoscope is applied, when the murmur can, in many cases, be made to appear and disappear by increasing or relaxing the digital pressure. In a certain proportion of cases it would appear that the right common carotid is abnormally near the surface. When this is the case we can understand how murmur is readily produced by comparatively slight pressure of the stethoscope. That it may be abnormally superficial is borne out by the fact that its pulsations can be very readily felt. In other cases the systolic murmur heard over the vessels in the neck is probably produced by the reflux wave from the right ventricle traversing a relatively constricted portion of the vein —the constriction depending upon the pressure of the stethoscope, or on the position in which the vein is placed by the position of the head and neck during auscultation.

Having thus stated what I believe to be the clinical facts regarding the so-called aortic and the arterial murmurs, it becomes of interest to briefly review some of the opinions which have been held as to their mode of production.

Hope and his school, as already referred to, held by the formula that deteriorated blood, unfilled vessels, and velocity of current were the causes of the murmur, both at the aortic orifice and in the course of the vessels.

Bouillaud held *bruit de souffle*, under all circumstances, to be due to increase of friction, but abandoned the attempt to explain it further.

Skoda entirely denied that velocity of current and watery blood were sufficient to cause murmur, and held that arterial murmur was due either to increase of friction or to vibrations of the vascular walls.

Hughes advocated deterioration in quality, increase in velocity, and, consequently, increased "attrition."

Beau advocated increase of friction, the result of want of proportion between the volume of the blood-wave and the channel it had to traverse, and denied that the vascular murmur was situated in the veins.

Bellingham adhered closely and intelligently to increase of friction as the cause of the murmur, and believed that the murmur at the orifice of the aorta was to be explained by diminished viscidity and increased rapidity. The same conditions were held to produce murmur in the arteries. In the veins he held that the murmur was produced by putting the vessel on the stretch, and then by gently pressing on it with the stethoscope.

Flint depended on abnormal changes in the blood for their explanation.

Sibson attributed the aortic murmur to lessened power and lessened contents. He did not understand the pulmonic murmur when it appeared early; but when it appeared late, and when the tricuspid murmur was disappearing, he thought it was due to lessened contents in the vessel.

Walshe placed the murmur at the aortic and pulmonary orifices; and, although he concedes that the condition of the blood must have an influence, he thinks the real mechanism obscure.

Hayden ascribed hæmic murmurs to increased friction in the blood-stream, and increased vibration of the vessels; and places the murmurs at the arterial orifices, and in the arteries and veins.

Niemeyer held that the cardiac murmurs in chlorosis were due to abnormal tension of valves and arterial walls, not to altered blood; while in the veins it was due to whirling eddies throwing the walls of the veins into sonorous vibrations.

Balfour revives Beau's theory of large blood-waves entering the aorta and pulmonary artery in the most fully-developed stages of chlorosis; while he follows Savart and other French observers in attributing the venous murmur to a fluid vein produced at certain permanently dilated parts along the venous tract.

The mode in which *bruit de souffle* was produced, especially in the vessels, gave rise to an enormous amount of controversy about the middle of the present century. It was at the same time much studied by the aid of physical experiments.

Corrigan was one of the first, if not the first, in this country, to investigate the phenomena by these means (*Dublin Medical Journal*, vol. x., 1836). He singled out Bertin's statement, that *bruit de souffle* was easily accounted for, where orifices are narrowed, by increased friction necessarily occurring under such circumstances. He argued against this, maintaining that the murmur was due, not to *increased*, but to *diminished* friction. He formulated thus the conditions which constitute its

mechanism: "1st, A current-like motion of the blood (instead of its natural equable movement), tending to produce corresponding vibrations in the sides of the cavities or arteries through which it is moving; and 2nd, A diminished tension of the parietes of the arteries or cavities themselves, in consequence of which their parietes are easily thrown into vibration by the irregular currents of the contained fluid, which vibrations cause, on the sense of touch *fremissement*, and on the sense of hearing *bruit de souffle*." The minuter details of this hypothesis he stated thus: "the particles in the centre being impelled with the greatest velocity, while those towards the sides are moving with varying or diminished velocities, whirling or currents are thus produced, which impinge upon the sides of the artery, and cause in them corresponding and extremely rapid vibrations;" and these vibrations are heard and felt (pp. 180-1). He was led from the foregoing to hold that other conditions, besides pressure or contraction, might give rise to the two conditions stated above as necessary to cause murmur, and gives as an example, amongst others, the arterial murmurs in aortic regurgitation. This same current-like motion, combined with lax arterial walls, were also considered the cause of murmurs after severe hæmorrhage. He gives the following explanation of the murmur heard in hysteric, nervous, or irritable patients —"in such persons the action of the heart and arteries is rendered irregular by very slight causes; an instant, although often momentary alteration in the motion of the blood, and a deviation from the natural state of tension of the arteries, is the immediate effect, and *bruit de souffle* is heard" (p. 191). It is unnecessary to reproduce here the experiments he performed to prove his position.

The whole structure falls to the ground when we find it supported by the contention that in aortic regurgitation there are no murmurs in distant vessels, as the femorals, for we know that pressure with the stethoscope sufficient to interfere with the calibre of the comparatively empty vessel, readily produces a double murmur, even in these vessels.

The London Committee of the British Association, in their report on "The Abnormal Sounds of the Heart and Arteries" (1837), came to the conclusion that "a certain resistance, or impediment to a liquid current, is the essential physical cause of all the murmurs produced by the motions of fluids in elastic tubes," and denied that the condition of the wall of the tube beyond the contraction had anything to do with the production of the murmur.

Corrigan replied to this (*Dublin Medical Journal*, vol. xiv., 1839), but his reply was mainly a repetition of his first paper.

Leared, in a paper (*Dublin Quarterly Medical Journal* vol. xiii., 1852), giving the results of his investigations into the sounds of the heart, says, that the "*bruit de souffle* is the result of a diminution, mutually exerted in the pressure of the particles of a body of fluid in motion. The consequence of this is, the main current becomes split into numerous smaller ones. The particles, in place of a progressive motion in which an equable relation to each other was preserved, assumed new and irregular movements" (p. 351). This enunciation of law was deduced from an experiment made by pumping fluid through a tube into a vessel full of fluid, the end of the tube being under the fluid, and then auscultating the end of the tube by having the bell end of the stethoscope under water and close to the orifice of the

tube. Any deductions as to the *bruit de souffle*, from such an experiment, seem to be entirely valueless, more especially as similar experiments led him to advocate views as to the causation of the cardiac first sound which have been abundantly proved to be erroneous.

Chauveau (*Gazette Medicale de Paris*, 1858), from a series of experiments, deduced the important proposition, that vascular murmurs do not depend directly either on the quantity or the quality of the blood. That they did not depend on the quality of the blood, he proved by exposing the cervical vessels in a horse, and auscultating them before and after repeated bleedings, until a very marked degree of anæmia was produced. He found that, with proper precautions as to the manner of auscultating, no murmur was present under these conditions. By injecting fluid into the circulation, and auscultating the vessels with the same care, he proved that a murmur was not produced by alterations in the quantity of the blood.

Next, by attaching an artificial aneurism of caoutchouc to the carotid artery, he showed that a *bruit de souffle* was produced in it; and he deduced from this, that, when a dilatation exists on the course of a vessel, a murmur is produced at that part. This generalisation is too sweeping, for there are many cases of aneurism in which there is no murmur produced.

He further showed, what of course had been known before, that any interference with the calibre of a vessel led to the production of murmur. By an ingenious series of experiments he showed that the murmur was not produced at the point of constriction, but in the part immediately beyond, where in fact there was a condition of relative dilatation.

He found that if the jugular vein was constricted in its

middle part no murmur was produced by the constriction; but that if it was constricted as it entered the chest a murmur was produced. He explained these as follows:—"In the first case no murmur was developed, because the vein exposed to, and yielding to atmospheric pressure, collapsed in proportion to the amount of constriction, and in this way a relative dilatation beyond the constriction was not obtained. In the second case the murmur was produced by a relative dilatation being obtained, for the dense fibro-cellular attachments of the lower part of the vein to the surrounding tissues prevented it from collapsing.

For the production of murmur it was also necessary that the difference between the diameter of the constricted part and that of the part beyond should be pronounced, and that the blood should flow with a certain force.

The mode in which *bruit de souffle* was produced was shown by Savart to be as follows:—"When the blood entered a part really or relatively dilated, it formed a fluid vein, which traversed the blood contained in the dilatation, and all fluid veins are the seat of vibrations which are susceptible of producing sound."

Using Savart's discovery, Chauveau's formula was as follows:—"*Bruit de souffle* is produced by the vibrations of the intravascular fluid vein, which always forms when the blood enters into a part really or relatively dilated of the circulatory apparatus" (p. 312). The physical conditions, briefly stated, were as follows:—"Whenever a liquid, impelled by a certain pressure, escaped from an orifice, it formed a fluid vein; the molecules which composed this vein vibrated and made the lips of the orifice through which it flowed vibrate; these vibrations produced murmurs similar to those in the vessels" (p. 312). These vibrations can be felt, as every clinician knows.

He held that the diminution in the quantity of the blood led to lowering of arterial tension. As a consequence of this, and of the diminished weight on the sigmoid valves, the heart drives the blood towards the arterial orifices with great energy. In anæmia he held there was a strong pulse with diminution of the volume of blood, and he supports this by appealing to some observations he made by means of the hæmodynamometer, which showed that the difference in arterial pressure during ventricular systole and diastole covered a wider range in anæmia than they did in health; in anæmia, the range being 14 centimetres, while in health there was only a range of 7 (p. 341). He noted this increase of force and of velocity in a high degree in animals rendered rapidly anæmic by large bleedings. The conditions, however, are so different in rapidly induced anæmia to what they are in anæmia produced more slowly, that experiments conducted by the former of these methods are of little value. Further, he bases his idea of increase of force on what appears to be a misconception of his hæmodynamometric observations. Range of movement cannot be accepted as the criterion of power. Sustained tension and a comparatively smaller wave denote greater power than the sudden and impetuous wave of considerable magnitude, followed by as sudden and as large a collapse. With the sphygmograph we often obtain this large wave line, but we do not regard it as an indication of increased power.

Although his experiments on vascular murmurs are thus most valuable, and it may be said conclusive, he did not adopt a like conclusive method in the examination of the murmurs heard over the precordia. He argued that they were produced at the arterial orifices; and their

mode of production was as follows: the ventricle adapts itself to the diminution in the volume of the blood, and in doing so, produces by its contraction a degree of contraction of the arterial orifices: the large arterial trunks do not, however, share in this contraction or become lessened in internal capacity; in this way, therefore, is produced that relative dilatation beyond the constricted part which is necessary for the production of murmur. He advances the ordinary arguments drawn from auscultation to support the view that the murmur has its seat at the arterial orifices. He grants that the constriction is slight, but this, he thinks, is compensated for by the "truly remarkable force" (p. 356) with which the blood leaps through the constriction.

When brought to the test of clinical observation, this explanation completely fails. The murmur is present when there is no special increase in the velocity or the force of the circulation at the arterial orifices. And pathologically it fails, for, as has been seen, the heart does not become smaller, with a view to adapt itself to any diminution in the total quantity of the blood; but, on the contrary, it becomes larger, and its chambers become more dilated, so that the condition which is supposed to produce a relative constriction at the arterial orifices does not exist.

He maintained that in anæmia there is no murmur in the arteries, but that a murmur is readily produced by the pressure of the stethoscope, as they are very *dépressibles*, and because of the velocity of the circulation. This agrees with the results obtained by careful clinical examination, and has been already referred to.

Marey ("Circulation du Sang," Paris, 1863) says, that although the conditions of existence of the *bruit de souffle*

are known, its immediate cause has not received a satisfactory solution. And he asks whether we are to accept Chauveau's teaching, or Heynsius'. The latter regarded it as due to movements of the nature of a *tourbillon* or eddy transmitted to the liquid by the current traversing it (p. 469).

He constructed an apparatus by means of which he showed that a fluid vein traversing fluid at rest produced a loud *bruit de souffle*, and also a vibration, analogous to what occurs in the circulation.

He thus summarises the conditions on which a *bruit de souffle* depends: a rapid current, which usually necessitates that a dilated part succeeds a narrowed part, and that the fluid impelled by a great force meets a feeble pressure in front of it (p. 471).

He further showed that the character of the murmur was affected by the calibre of the mouth of the tube beyond the constriction. From this he held that the production of the murmur depends on the degree of tension on the two sides of the constriction: the larger the exit-opening of the tube, the lower the tension on that side of the constriction, and *vice versa;* and the lower the tension, the greater the rapidity of the current through the constricted part (p. 474 *et seq.*).

In anæmia and chlorosis he likewise held that there was no arterial murmur, but that it was readily produced by the pressure of the stethoscope; and he ascribed the ease with which it is produced to low arterial tension, and the energy of the arterial pulsation (p. 476). The murmur produced at the orifice of the aorta in chlorosis, fever, &c., is to be explained, he held, on this same principle of lowered arterial tension. The low tension he ascribed to a watery state of the blood, which allowed it to pass more

readily through the capillaries, and probably to the dilatation of the capillaries making the channels for the blood larger. He held that in the normal state the tension in the aorta is sufficient to prevent the fluid vein shot into the vessel during ventricular systole from vibrating; but that, after bleeding, the tension is lowered so much, and the rapidity of the stream is so increased, that a *bruit de souffle* is produced (479 *et seq.*). Here, again, we see rapidity of stream insisted upon as a condition, but this does not agree with clinical observations. And lowered tension and rapidity of stream do not meet the necessities of the case in the pulmonary artery.

Bergeon produced a most valuable thesis in 1868 ("Causes et Méchanisme du Bruit de Souffle." Paris, 1868), confirming many of Chauveau's views. He further worked up some minor points bearing upon the facility with which different fluids produced murmur, and also the conditions of rapidity and tension which affect the development of murmurs. But his main conclusions so resemble those which have been already given in considerable detail, that it is unnecessary to consider them at length.

In conclusion I may briefly summarise my position as follows:—

1. The murmur which in chlorosis, &c., is regarded as basic, and as being produced at the aortic orifice, is really a tricuspid regurgitant murmur. This explanation alone meets the facts obtained by careful auscultation, and by a complete study of the state of the circulation in these conditions.

2. There is no arterial murmur although one is readily produced by constricting the vessel by the stethoscope, &c.

3. The venous murmur is produced in the veins at the root of the neck by the pressure of the stethoscope, or by

a localised interference with their calibre by putting the parts attached to them on the stretch.

4. The continuous venous murmur is produced by the continuous venous current passing the constricted part of the vein, and its accentuations are due to a reflux wave produced by ventricular or auricular systole, or by both, passing the constriction.

5. The quality of the blood is a factor in the facility with which murmurs are produced, but it is not sufficient of itself to cause murmur, a greater or less degree of constriction in the course of the vessels being the essential element.

CHAPTER V.

SOME POINTS CONNECTED WITH TRICUSPID MURMUR AND MITRAL STENOSIS.

THE following cases and observations are offered in elucidation of some points connected with tricuspid murmur, and the position of the heart in mitral stenosis. At the same time, they are intended to support the contentions in chapter iii. as to the site of the principal cardiac murmur in debility.

In mitral stenosis it has been taught, and is still held by some, that the pulsation above the fourth rib to the left of the sternum is due to auricular enlargement. Now, it is quite true that pulsation in this region is often associated with dilatation of the left auricle, but there is, along with the auricular dilatation, a dilatation and hypertrophy of the right ventricle. When the right ventricle is thus affected as the result of mitral lesion, it extends to the left of its normal position, and produces in the third space, and sometimes in the second also, the pulsation referred to by some authorities as auricular. In chapter iii. it has already been shown that this extension of the right heart to the left buries the left auricular appendix so deeply in

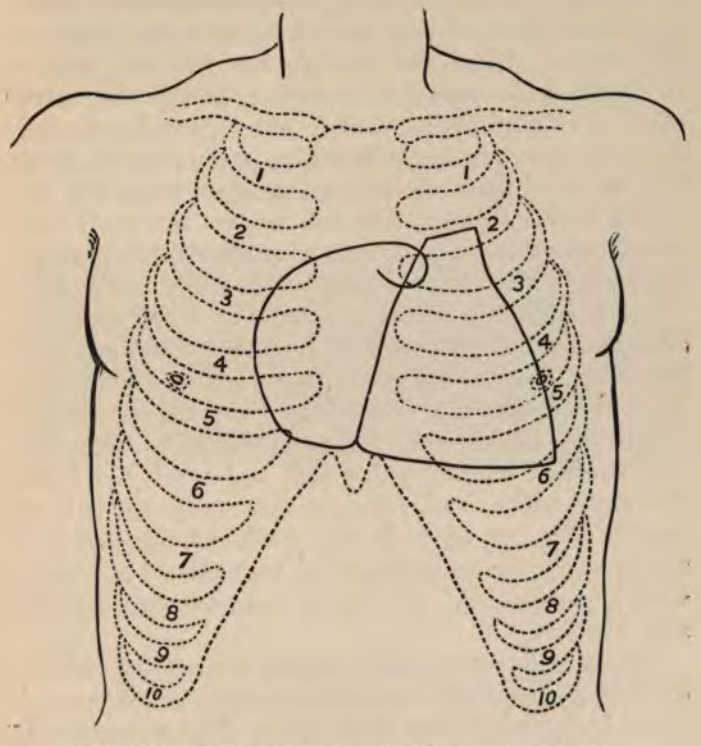
the chest that it is quite impossible for it to approach the chest wall even as closely as it normally does. It therefore follows that to regard the pulsation or dulness in any of the left spaces as due either to the auricle or its appendix is not consistent with fact.

The following case is a valuable and instructive one, as marking the area over which a tricuspid murmur may be audible, and as showing what the relations of the heart are to the thoracic wall:—

CASE XIV.—Mrs. H., æt. 21, had been confined to bed for weeks with cough and shortness of breath. Her nights were restless, for cough and dyspnœa prevented her lying down. There were râles in both lungs. There was a loud, rough-toned systolic murmur over the precordia. On more precise observations being made it was found that the inferior and left limit of this murmur was in the nipple line; while to the left of this there was a soft and faint systolic murmur heard towards the axilla, and which gave the impression, as soon as the nipple line was crossed, of being conveyed from a distance. From the nipple line inwards (to the right) the murmur maintained the same rough-toned character as high as the second left space, in which space it was heard for one to two inches from the sternum. It presented the same character as high on the sternum as the second costal cartilage, while above this it was a soft murmur. It was also heard for a short distance in the second right space, and for a greater distance in the third and fourth spaces on the same side. There was no murmur in the carotids. There was no apex thrill, no cardiac irregularity, no reduplication of the second sound, and no presystolic murmur. There was pulsation in the second, third and fourth left spaces; in the second for one to two inches

from the sternum. The veins over the upper part of the sternum were not only distended but pulsated synchronously with ventricular systole. Whenever the patient lay down cough became severe, and she was compelled to sit up again. She improved greatly under treatment; the cough and dyspnœa disappeared, and all her former discomfort. The treatment consisted of cardiac tonics, mainly digitalis. Under the use of the latter the area of audition of the murmur became more circumscribed: it became inaudible over the manubrium and over part of the area to the right of the sternum, while at the same time it had increased in loudness over the lower part of the sternum, and between it and the nipple. The murmur extended in area whenever the digitalis was withdrawn. The veins over the sternum lost their pulse and their distension. This patient was under observation for fully two months, during which period her condition continued satisfactory, but the symptoms tended to return whenever cardiac tonic treatment was long suspended. She was delivered of twins about two months after I first saw her. As soon as labour pains came on, the heart began to show signs of faltering; I therefore delivered her without delay, and without difficulty, by means of the forceps. By the time that this was effected she was almost pulseless, livid, and semi-unconscious; she, however, rallied completely under the influence of ether administered hypodermically. She died some days afterwards of a form of puerperal tetanus. At the *autopsy* careful observations were made on the position of the heart as it lay *in situ*, my friend Dr. Lockie of Carlisle being present and verifying my observations. The entire anterior aspect of the heart was made up of right ventricle and right auricle. The origin of the pulmonary artery was at the level of the upper border of the

second left rib. The conus arteriosus extended from one to two inches to the left of the sternum in the second space; the inner part of the space being occupied by the



CASE XIV.—Showing the position of the heart after death.

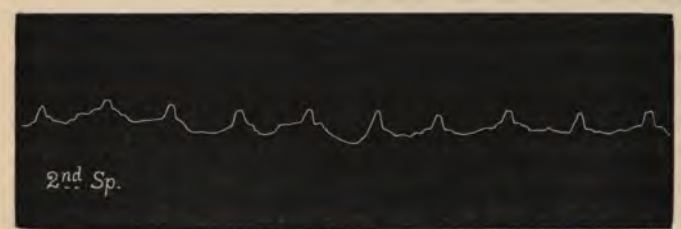
auricular appendix of the right auricle. The left auricle and its appendix were deeply buried in the thorax, and were quite invisible from the front. The right auricle

extended into the second, third, and fourth right spaces, as seen in the annexed diagram. The mitral cusps, which were much thickened, formed by their union a fibrous ring which only admitted the index finger as far as the second phalanx. An interesting observation was made during the process of injecting the left ventricle from the aorta with water. When the ventricle was full fluid readily regurgitated in a considerable stream through the mitral valve into the auricle; but when the tension in the ventricle was raised, by hyperdistending it, the pressure of the fluid so acted upon the valves that they completely prevented further regurgitation, and became absolutely competent. The tricuspid segments were also adherent to each other and thickened, producing a degree of stenosis which only admitted the tips of two fingers. When water was injected into the right ventricle from the pulmonary artery the tricuspid valve was found to be largely incompetent, the centre of the valve presenting an opening the size and shape of a small almond when the ventricle was full. The right auricle was lined with a pale and tough blood-clot. The right auricle and ventricle were proportionally enlarged. The left ventricle was small, and the auricle of the same side was enlarged, being both dilated and somewhat hypertrophied. The aortic and pulmonary arteries were healthy.

This case illustrates very perfectly the area of audition of a tricuspid regurgitant murmur, due to incompetence produced by organic disease of the valve. This area extended from the nipple line inwards over a triangular area, the left boundary of which was formed by a line drawn from below the nipple to a point in the second left space one to two inches from the edge of the sternum, over the sternum adjoining this area, and also into the second, third and

fourth right spaces. It therefore exactly coincided with the position occupied by the right heart as seen after death, and is what would *a priori* be expected. Such a case quite controverts the old opinion that the right heart is too feeble to declare its power by sound, or its valvular imperfections by murmur.

The pulsation in the second and third left spaces was seen to be caused by the right ventricle. The pulsation in the second space was traced, and is given below, as it is an almost exact counterpart of one taken by my friend Dr. Gibson (*Edinburgh Medical Journal*, October, 1877)



CASE XIV.—Tracing of pulsation in second left space.

for Dr. Balfour when he was resident in his wards, and which has been held by Dr. Balfour to confirm his teaching that this pulsation is due to the left auricle, or to its appendix.

The tracing is of very little value, as the interspaces were narrow, and the heart was not as fully in contact with the chest wall as it sometimes is. It, however, shows the period of the filling of the ventricles and the systolic impulse.

The presence in this case of a systolic murmur over the manubrium, and its disappearance under a plan of treatment which led to considerable rehabilitation of the heart,

as evidenced by diminished distension of the right side and of the venous system, show that the murmur was audible there not as the result of its propagation from the lower part of the sternum, but of a greater degree of dilatation expanding the right heart more upwards. The murmur became inaudible and continued so, although the murmur over the lower part of the sternum had become louder, and the power of the right ventricle had increased. Had the murmur been simply conducted up the sternum, it would either have continued audible, or have increased in loudness in proportion to its loudness at the lower part of the sternum.

The faint and distant systolic murmur heard beyond the apex towards the axilla was, I believe, a mitral regurgitant one, for I do not believe that tricuspid murmurs are heard beyond the cardiac area proper, save when they are heard over the liver owing to the enlargement of that organ, and to the degree in which the right heart comes into contact with it. The proof of this statement is to be found in such cases as the preceding is an instance of. In it the murmur was not always propagated to the manubrium sterni, and never to the right of what proved to be the limit of the right auricle, while the physical properties of the sternum would specially lend themselves to this conduction were the propagation of murmurs so universal in direction, or, it may be said, so diffuse in their behaviour, as is frequently assumed. In this case physical examination during life showed that there was undoubtedly mitral reflux. The conduction of the murmur towards the axilla was proof of this, while the faintness of the murmur was due to the small volume of blood in the ventricle and the proportionally small quantity which could regurgitate. It was, however, also shown after death by injecting the

ventricle from the aorta; for the mitral valve only became competent when there was a fulness and a pressure within the ventricle which it could not have developed in life, seeing the degree of stenosis was such as would only permit of an imperfect supply of blood to it.

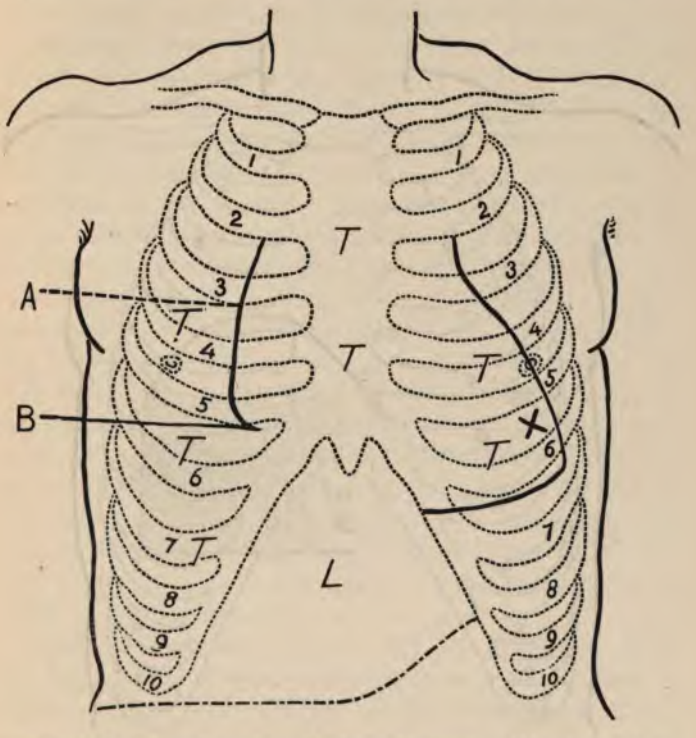
Increase of intra-ventricular pressure closed the orifice by mechanically forcing the hard and thickened valve into a position which it did not assume without the aid of this pressure. The valve, when forced towards the auricle, was competent; when not in that position it was incompetent. In cases of great stenosis the rigid valve tends to be directed towards the ventricle, and as the suction and the propelling power of the ventricle diminish, it must become impossible for the ventricle to thrust the valve sufficiently towards the auricle to close the orifice.

The following cases may be given to illustrate some of the variations in the area of audition of tricuspid murmurs:—

Case XV.—Benjamin B., æt. 28, an engineer, was admitted into hospital on the 30th of July. He had had rheumatic fever before Christmas, at which time he had been confined to bed for ten weeks. He returned to work, but had again to abandon it, owing to shortness of breath and palpitation. On admission, respiration was evidently accelerated, and there was a diffused pulsatory movement in the head and neck. The external jugular veins were markedly distended. The cardiac impulse was diffuse, its maximum impulse being visible in the fifth left space, four inches from midsternum. There was a thrill over the apex, and the cardiac action was irregular in force and rhythm. On auscultation, the first sound at the normal site of the apex was thumping, and accompanied by a faint systolic murmur, which conveyed the idea of being

distant, and was quite lost to the left of the limit of cardiac dulness, and was not heard in the axilla or at the angle of the left scapula. On moving towards the sternum the systolic murmur became more audible, and no longer gave the impression of distance. The murmur was heard as high as the second left space, and over the sternum to the level of the second rib, but was quite inaudible over the manubrium. It was also audible in the second right space, and towards the right over the whole area of hepatic dulness, as seen in the diagram, and extended as far to the right as the posterior axillary line. The area of audition of the murmur is marked T in the diagram. The comparative liver dulness began at the level of the third rib, and this line bounded the upper limit of the murmur. Its lower margin was felt in the abdomen nearly at the level of the umbilicus. The urine contained no albumen, its specific gravity was 1015, and he passed from 50 to 60 ounces in the twenty-four hours. There were no râles in the lungs. On 5th August, the cardiac action had become quieter and more regular, and at the apex there was an inconstant systolic murmur heard only every few beats. The second sound was much accentuated and also reduplicated. In the third left space, about two inches from the sternum, there was a very marked pulsation. When a flag was placed over this point, and another over the pulsation in the fifth space, inside the nipple line, the two flags moved synchronously: when one was transferred to the outer area of pulsation in the fifth space, the flags distinctly followed each other, that is to say, the flags in the fifth space did not move synchronously. Under rest and medicinal treatment this patient's condition improved greatly. The area of audition of the murmur

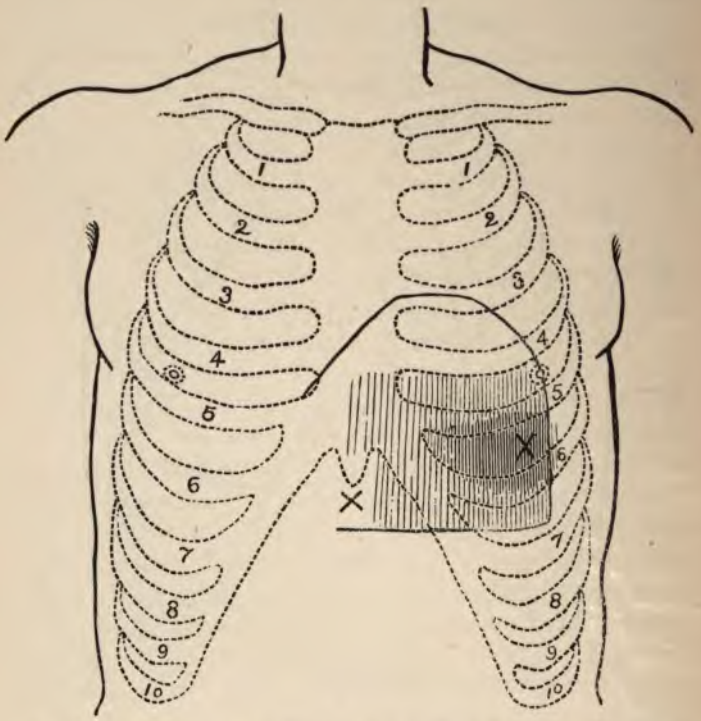
gradually diminished, until, to the right of the sternum, it could only be heard as a very faint and distant



CASE XV.—T marks position of tricuspid murmur. X is over maximum cardiac pulsation. A marks upper limit of comparative liver dulness. B marks upper limit of absolute liver dulness L is over the liver dulness in the epigastrium.

and softly blowing sound. He left hospital and was lost sight of.

CASE XVI.—Mrs. E., æt. 40, had suffered from rheumatic fever seven years previously to my seeing her: the attack was severe, and she was ill for sixteen weeks.



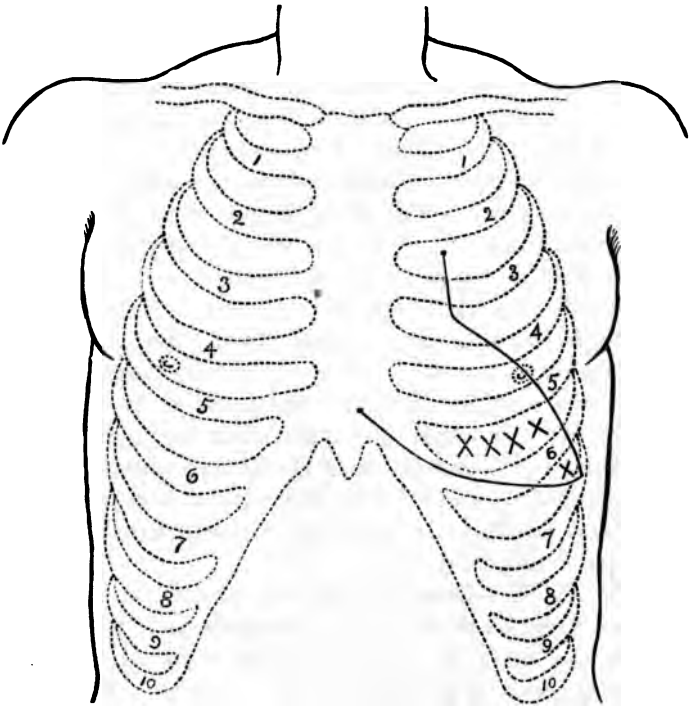
CASE XVI.—X marks site of pulsation. Vertical lines mark area of murmur.

Occasionally she "caught cold," and had a cough; her feet also swelled during these attacks. She was suffering from such an attack when I saw her. The pulse was 92,

regular, and small. Cough was always worse when she lay down. The area of cardiac dulness is seen in the preceding diagram. It commenced at the third left rib, and extended on that rib for $2\frac{1}{4}$ inches from midsternum; in the fourth space it was $3\frac{3}{4}$ inches from the same line; and in the sixth space, $4\frac{1}{2}$ inches; while to the right it was only 1 inch from midsternum. There was pulsation over the large veins in the neck. There was also pulsation in the fifth left space, and in the epigastrium, as marked X in the diagram. There was a systolic murmur in the fifth left space, loudest over the maximum pulsation in that space, but audible along that space to the sternum, over a small portion of which it was also audible. It was also audible in the adjoining epigastrium, and slightly in the fourth and sixth spaces, and was also propagated slightly to the left. In this case the murmur was low in situation because the heart was; but the murmur was heard over what would have been found to be the right side of the heart had the opportunity been afforded of examining it. The relation of the area of murmur to the area of dulness, coupled with the signs of venous distension, leave no doubt in my mind that this murmur was a tricuspid regurgitant one.

Case XVII.—Owen C., æt. 66, also illustrates how the heart, by its displacement outwards and downwards, affects the area of audition of the tricuspid murmur. He complained of palpitation and weakness. The pulse was 88, regular, but rather small and weak. The vessels were hard. There was a trace of albumen in the urine, and there was a history of nocturnal calls to micturate. The thorax was barrel-shaped. It rose and fell during respiration, but expanded only slightly. The cardiac dulness is outlined in the annexed diagram. Such an

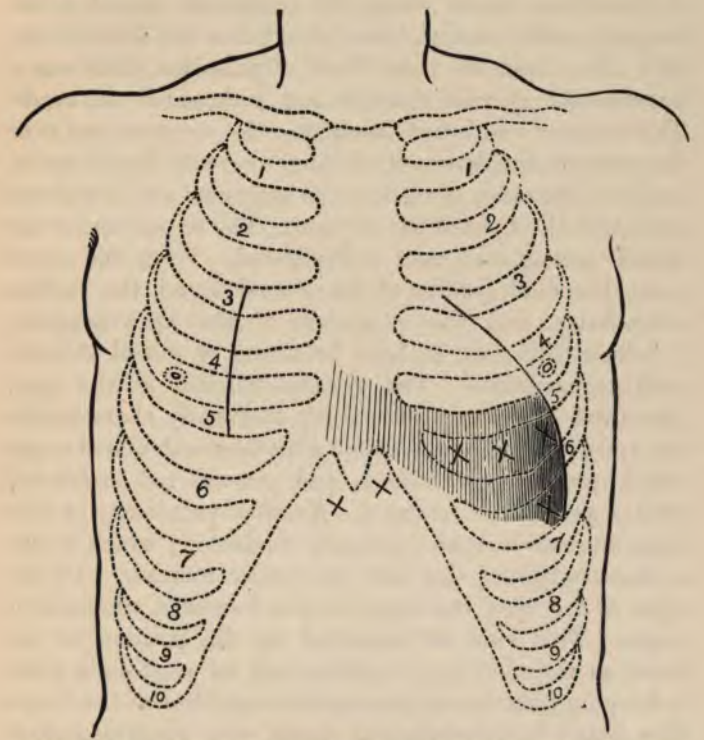
area of dulness as this made me at one time doubt the accuracy of my percussion; but I have so frequently submitted it to the test of *post-mortem* observations with satisfactory results, and have also been so sur-



CASE XVII.—Showing area of cardiac dulness. Xs over seat of impulse.

prised at the variation in the position of the heart, that I am not now sceptical of the accuracy of the results obtained by this method of investigation. There was pulsation in the fifth and sixth spaces, marked with

Xs in the diagram. In the fifth space, along the line of pulsation, a blowing systolic murmur was on one or two occasions audible after exertion, but it was frequently absent.



CASE XVIII.—Xs over pulsation. Vertical lines show area of murmur.

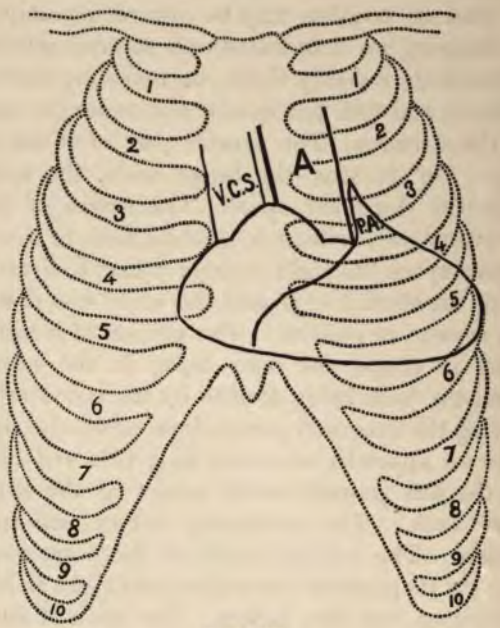
CASE XVIII.—An old soldier, æt. 46. The pulse was 76, irregular, and very small, being little more than a thread. The chest was barrel-shaped, and expanded little

during respiration. There was no fulness or pulsation of the veins of the neck, but the veins over the upper part of the thorax were abnormally distended. The apex beat was in the sixth space, about an inch outside the nipple line. Pulsation was visible where the crosses are placed in the diagram, and is seen to have been below the level of the fifth rib. Over the outer limit of pulsation there was a harsh-toned systolic murmur not propagated outwards. The murmur was heard inwards to the sternum and over the sternum, not, however, reaching above the fourth space, or above the same level over the sternum; and it was not audible to the right of the sternum. The second sound was closed, accentuated, and reduplicated. From the small pulse, the reduplication of the second sound, the cardiac irregularity, and the roughness of the apex-murmur, I believe this case to have been one of mitral stenosis with regurgitation. The systolic murmur at the apex presented the rough tone which frequently characterises the systolic murmur associated with those valvular changes which produce constriction, and yet are not associated with a presystolic murmur. Another peculiarity of this apex murmur is, that it is badly conducted; which is also a characteristic of the true presystolic murmur. To the right of the apex the murmur was, I believe, tricuspid in origin. This view is supported by the position of the heart as revealed by percussion, and its position is quite in keeping with the emphysematous condition of the lungs. The heart, in barrel-shaped chests with emphysematous lungs, always occupies a lower position than it normally does. By this means, more room seems to be given to the large venous trunks within the thorax, for it is common to find in such cases no venous pulse, although the right chambers of the heart be distended.

CASE XIX.—F. J. M., female, æt. 58, died of exhaustion consequent on pelvic malignant disease. Before death careful examination had been made in the ordinary positions for cardiac murmur, but with a negative result. The heart was examined *in situ*, and illustrated in a very beautiful manner how its position may be altered, possibly by prolonged dragging on debilitated and relaxed attachments. The pericardium was only visible, on removing the sternum, in the fourth and fifth left spaces, and under the adjoining part of the sternum. The greater part of it was covered by lung. On drawing the lungs aside, the aorta was visible in the third left space. The origin of the pulmonary artery was, as seen in the diagram, in the middle of the same space, its right margin being half an inch to the left of the sternal edge, and the vessel was one inch in breadth, as seen in position. The portion of it visible was pyramidal in shape, the apex being in the second left space, and the base being formed by the conus arteriosus. On drawing the lung and pericardium aside, the tip of the left auricular appendix was seen as a thin red line lying against the left postero-lateral aspect of the artery for nearly an inch. The pulmonary artery was crumpled transversely. The inferior border of the heart was in the fifth space. Its greatest dimension from right to left, as it lay in position, was five inches. The apex of the heart was against the ribs in the midaxillary line. The arch of the aorta was straightened out as if by the dragging of the heart upon it; three inches of its ascending course were visible at one time. It will be thus seen that the heart lay much to the left and below its natural position, so that auscultation in the ordinary areas was absolutely valueless. Even had there been valvular imperfection, the murmurs would probably not have been heard through the depth of

lung which interposed between the heart and the thoracic wall.

The foregoing cases illustrate the position of the heart and the varying extent of the audition of the murmur



CASE XIX.
Position of heart after death.
V.C.S. Superior Vena Cava.
A. Aorta.
P.A. Pulmonary Artery.

due to tricuspid regurgitation. In Case I. the limits of the murmur exactly coincided with the limits of the cardiac murmur in advanced chlorosis. This strengthens the contention that in chlorosis the murmur is due

to a like regurgitation, although of a recoverable nature.

The limited area over which a loud presystolic murmur is frequently audible ought, I think, to influence our views as to the conduction of murmurs in a manner in which it does not appear to do. Were sound readily or ordinarily conducted through chambers occupying different sides of the heart, we should expect the presystolic murmur to be heard towards the sternum, for the presystolic stream producing the murmur impinges on the apex of the left ventricle, and also upon the septum, at this point. Further, the murmur is frequently so loud, that we should expect it to be propagated to the right, and this we know it is not. It would seem from this that there ought to be an *à priori* suspicion that systolic murmurs audible to the right of the apex are not so commonly due to mitral regurgitation as is supposed. I have several times noted a systolic murmur, audible at the apex, and propagated towards the axilla, and yet was quite lost to the right of what was legitimately assumed to be the septum of the ventricle. We must, therefore, either formulate our position by such observations, or conclude that physical laws are subject to inscrutable vagaries.

The degree in which systolic mitral murmur is propagated to the left and is heard in the axilla or at the angle of the scapula, depends upon the distension of the left ventricle and auricle bringing the former into closer proximity with the ribs laterally and posteriorly. This is further proved by the fact that the harsh systolic murmur which, as has been already said, often accompanies mitral constriction is not propagated much to the left of the cardiac dulness, as in such cases there is not that dilatation of the ventricle which accom-

panies and is necessary to the extensively propagated murmur.

The variability in the presence of the presystolic murmur is due, amongst other causes, to the varying distension of the right heart; for, as soon as failure of compensation manifests itself, the right ventricle becomes more distended, and with its distension the left ventricle is removed from the chest wall and acquires a deeper position. In this way the right ventricle covers the left, so that the murmur which, carried by the stream, and impinging at the apex, had communicated its sonorous vibrations to the thorax, and thence to the ear, no longer does so.

Proof is obtained from the clinical facts of mitral stenosis of the important part played by the aspiratory action of the ventricles in the maintenance of the circulation. In stenosis a time comes when there is failure of compensation, and the balance of the circulation is disturbed. This occurs in cases where the valvular changes have been long in existence, and where, from their nature, they cannot have advanced, or been materially altered, for a long period anterior to death. In such cases, I believe the failure in the aspiratory action of the ventricle to be responsible for the break down. It is not, as is taught by some authorities, due to yielding of the right ventricle producing lowered pulmonary tension, for the pulmonary circulation gives evidence of its continued tension in the accentuation of its second sound. But further, were it due to tricuspid failure, it may be asked, how comes the pulmonic congestion about? if the left heart be insufficiently supplied with blood, how come the pulmonary-capillaries to be so distended that effusion occurs into the alveoli of the lungs? The failure of the right heart is the symptom of a graver failure on the left side:

the break down is not the result of the right-sided failure.

Further, I do not believe that the left auricle plays such an important part in the circulation that the loss of balance is due to its debility; it may aid, but the important failure is to be attributed to the ventricle.

When the ventricle becomes weakened, it often attempts to compensate for it by increased rapidity. This increase is not to be always discovered by examination of the pulse, but by the increase in the rapidity of precordial pulsation. One of the last cases of mitral stenosis which I had under my care, and which had, under treatment, recovered from several previous attacks of cardiac failure, presented during the final loss of balance a very small, irregular pulse, beating between 50 and 60, while the cardiac pulsations were about 120. In such a case the precordial pulsation is produced by the right ventricle. The left ventricle probably continues to act synchronously with the right, although the small amount of blood in it, coupled, probably, with its weakness, leads to an imperceptible pulse. This is the explanation of the want of proportion between impulse and pulse, which so much puzzled some of the earlier observers. As has already been seen, the above explanation was first offered by Adams.

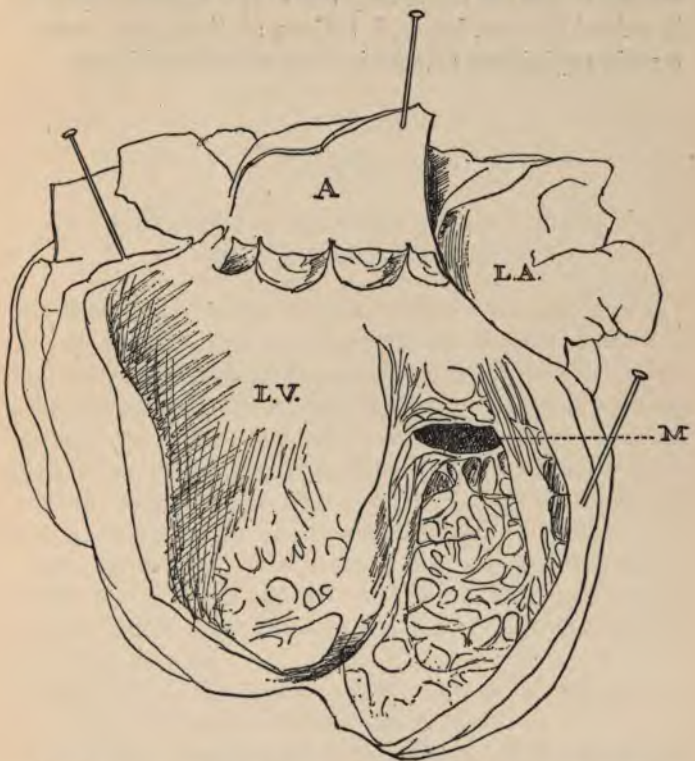
The disparaging estimate usually formed of the right heart is entirely erroneous, and the common habit of regarding the first sound of the heart, wherever heard, as the measure of cardiac power, is a fruitful cause of grave error in diagnosis and prognosis, even at the present time; and mitral stenosis is one of the conditions in which this misapprehension prevails. Fortunately for the safety of the lungs the right heart readily adapts itself to obstruction on the left side; but the failure of the circulation is

not due to deficient blood supply in the pulmonary circuit and left auricle. On the contrary, there is an abundant reservoir in both, were the suction power of the ventricle sufficient to take advantage of it.

Another interesting and instructive point is the posture assumed by persons who are the subjects of mitral stenosis. One of the first evidences of failure of compensation in this special lesion is to be found in the attitude assumed by the patient. He no longer lies down, but constantly occupies a sitting posture, for, as soon as the recumbent posture is attempted, cough and increased dyspnœa supervene. The explanation of this is to be found in the anatomical relations of the left auricle and ventricle. In the adjoining illustration, taken from the heart of Case I., this is well seen. The constricted mitral orifice M may be said to occupy the posterior segment of the roof of the left ventricle, so that in the sitting posture the flow of blood into the ventricle from the auricle is aided by the considerable influence exerted by gravity; for there can be no doubt that, as the aspiratory action of the ventricle is lost, as a result of the degeneration of its muscle, the influence of gravity becomes an important factor in the maintenance of the circulation by the assistance it gives in increasing the volume of blood in the ventricle. This influence is removed as soon as the patient lies down, which further implies that the relief to the auricle and to the pulmonary circuit is lessened. But, in addition, there is in the recumbent posture an increase in the facility with which regurgitation takes place from the ventricle. It is well known that mitral stenosis is almost invariably accompanied by regurgitation, and it can be easily understood that the horizontal position favours this backward flow of blood from the ventricle, and that this back-wave increas-

ing the capillary fulness is a second factor in producing cough and dyspnœa.

I have notes of an interesting case of this kind, in a



Mitral Stenosis—showing position of stenosed orifice, M; A, aorta; L A, left auricle; L V, Left ventricle.

woman in whom there was present, in the sitting posture, a rough systolic murmur at the apex as well as other evidence which led me to believe she had mitral stenosis.

On lying down cough at once came on, and a systolic murmur appeared over the right heart.

The mechanical effect here explained as dependent upon change of posture is not only one of the causes of dyspnœa in mitral disease, but is, I believe, at times, the cause of certain anginiform attacks in fatty and dilated heart.

CHAPTER VI.

THE DIFFERENTIAL DIAGNOSIS OF VEGETATIVE OR VERRUCOSE ENDOCARDITIS.

THE form of endocarditis associated with the presence of vegetations is so comparatively common that it is desirable to have an accurate knowledge of its morbid anatomy; but it is also desirable so to define, if possible, the symptoms and the evidence of its presence that its detection may be possible during life. Such knowledge not only aids prognosis, but we hope it will be seen that it even assists in the selection of remedial measures.

In approaching the subject it is necessary to consider briefly the other forms of endocardial inflammation, in order to define more precisely the place which the vegetative form occupies.

Ideas as to the pathology of endocardial vegetations have passed through various phases.

Corvisart ("Maladies du Cœur," Paris, 1806) maintained they were of syphilitic origin, compared them to venereal warts, and believed in the benefit derived from the anti-syphilitic treatment of them (p. 220 *et seq.*).

Laennec thought they were polypous or fibrinous concretions.

Hope agreed with Kreysig, Bertin, and Bouillaud, that they were due to inflammation (*loc. cit.* p. 368 *et seq.*).

Virchow demonstrated that there was "no exudation, but that the cellular elements take up a greater quantity of material, and the spot becomes uneven and rugged," and, the process proceeding, an "excrescence or condyloma" arose ("Cellular Pathology," 1858, London, 1860, p. 363).

Wilks and Moxon ("Pathological Anatomy," London, 1875) divide endocarditis into plastic and ulcerative, the former forming bead-like elevations by a true inflammatory process, the latter being a "rare and formidable complication" of the former, and leading to a loss of substance and aneurism of the affected valve.

Jones and Sieveking ("Pathological Anatomy," London, 1875) hold similar views as to their inflammatory origin, and that they may go on to ulceration leading to aneurism locally, and pyæmia from systemic infection; but they refer also to a "chronic ulcerative endocarditis" which has symptoms resembling chronic pyæmia.

Latham (*loc. cit.* p. 66) noted the fever, the pain referable to the heart, "the excessive impulse, intermittent, irregular, faltering, fluttering action of the heart," and the bellows murmur, as accompaniments or evidence of endocardial inflammation. The bellows murmur he considered an infallible sign.

Rosenstein (Ziemssen's "Cyclopædia of the Practice of Medicine," vol. vi.) adopts an admirable division of endocarditis. He divides it into three forms—(1) acute, diphtheritic, or ulcerative; (2) subacute, verrucose, or vegetative; (3) chronic, contracting, or sclerotic. The first, he says, "is distinguished no less by its acute malignant course, than by its anatomical products" (p. 64),

and he confines the term to those cases where a true ulcer is present.

Senhouse Kirkes was the first in this country (on the Continent Virchow claims precedence) to work out the relation between inflammatory disease of the endocardium and embolism in the brain and other organs. He also taught that the molecular debris from the seat of the inflammation, being taken up by the blood-stream, adulterated the blood, and gave rise to typhoid symptoms.

Walshe's account ("Diseases of the Heart," 4th ed.) of the evidence of the presence of acute endocarditis is too indefinite to enable any one to do more than, as a rule, guess the presence of the lesion, while in chronic endocarditis the only symptoms present are referred by him to the valvular lesion.

Trousseau ("Clinical Medicine," New Sydenham Society's translation, vol. iv.), in an exhaustive lecture on acute rheumatism and its sequelæ, confines the term ulcerative endocarditis to a very severe form of disease where there is a distinct ulcer, from loss of substance, on the endocardium. The disease was, in his experience, frequently ushered in by rigor. It was accompanied by prostration, diarrhœa, and other symptoms which might lead to the belief that the case was one of typhoid fever. The disease ended fatally from the 8th to the 15th day. He based the diagnosis on the exclusion of typhoid fever as the cause of the general symptoms, and on the presence of an endocardial murmur.

Flint (*loc. cit.*) thinks inflammation of the endocardium may result in exudation of lymph below and upon the membrane, as well as in deposit of fibrin. When endocarditis is developed he thinks there may be no symptoms to attract attention, or they may be so incorporated with

the rheumatic affection usually accompanying the disease as to be inseparable from it. The condition might probably give rise to febrile movement if found alone; but otherwise the febrile movement could not be distinguished from that caused by the rheumatism (p. 390 *et seq.*).

Hayden (*loc. cit.*) says the symptoms of acute endocarditis are "by no means characteristic" (p. 802). He relied much on the presence of murmur, and says the pulse is quickened, and that discomfort at the region of the heart may be complained of. He warns against confounding the murmur with the hæmic murmur, but offers little to guide us in distinguishing between it and the "dynamic" apex murmur. The supervention of acute endocarditis on old standing affection of the valves he regards as scarcely recognisable.

In such an admirable and valuable text-book as Bristowe's "Theory and Practice of Medicine," which is in the hands of many of the practitioners of medicine, there is little to guide the student towards the recognition of endocardial inflammations. He says the symptoms are neither "striking nor serious;" a murmur may be present, and a new murmur may be developed. There may be uneasiness and pain, but the indications are "slight and fallacious." He further adds that "from the smallness of the extent of the inflamed surface we should scarcely expect much febrile disturbance to be evoked; nor, as a rule, is simple endocarditis attended with marked fever. Still there may be elevation of temperature, thirst, scanty urine, and other indications of the febrile condition," and some excitement or modification of the action of the heart.

At medical societies specimens of "ulcerative endocarditis" are frequently shown which, from the recorded descriptions, appear to be specimens of endocardial vegeta-

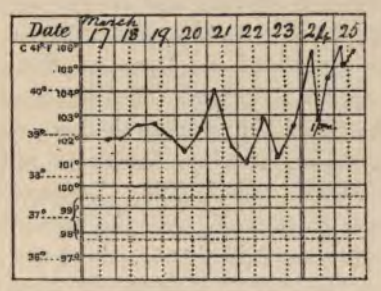
tions. It would indeed appear that the term ulcerative is commonly applied to these vegetations, and that they are regarded more as objects of pathological interest than as the result of an affection which it is the duty of the physician to recognise.

At the extreme limits of endocarditis are phases of the affection which vary vastly in intensity and in results. At the more benign limit is the chronic or sclerotic form. This condition is slowly produced, and may, in its origin and course, be more justly described as a prolonged irritation, with slow hyperplasia of the fibrous tissue of the part, than as an inflammation. It frequently dates its onset from an attack of rheumatic fever, but it also occurs in persons of a rheumatic or gouty diathesis who may never have developed either disease in a typical form. It is frequently associated with kidney cirrhosis, and may even be regarded as one of the natural changes slowly supervening with advancing years, for it is very commonly present, in a greater or less degree, in the hearts of old persons. The condition may be more or less active in its onset, leading early to a considerable swelling of the valve, but it never leads to abrasion of the endocardium or to the precipitation of fibrin upon it. Its origin must often, if not invariably, be unaccompanied by any symptoms leading to its detection, and its progress continues as the causes which led to its initiation continue to exist. Its results are purely mechanical, and such as accrue from valvular imperfections.

At the other extreme are found cases of true ulcerative endocarditis, which are either primary, or, on the other hand, are secondary to some infective disease, as puerperal fever. In this class there is a very acute inflammatory process, usually localised to a small area, rapidly going on

to destruction of the part affected, and ending in the products of the destruction being carried away by, and circulating with, the blood. There is a true ulcer formed from loss of substance, and vegetations not unfrequently surround the ulcerated area. The ulcer may lead to perforation or to aneurism of the valve affected. When this acute endocardial affection is the result, or one of the local manifestations, of an infective disease, it may in its turn become the source of infection to other organs by the transmission of infective emboli, which may even produce abscesses in the organ in which they become arrested. The acuteness and character of the local affection when associated with a primary infective lesion is probably due, in part at least, to an inflammation of a necrotic type, in which the infective factor (probably an organism) has rapidly developed, and whence it can again gain ready access to the blood when the necrotic condition has sufficiently advanced. But it would appear that the disease may assume an almost equally rapid course, although not associated with any infective lesion, and certainly without developing secondary pyæmic foci. When the endocardial lesion is thus primary, it is probably simply an acute form of inflammation rapidly producing a condition analogous to that of acute abscess formation in other tissues, and is probably not a true infective process although micrococci may be present, as in acute abscesses elsewhere. But the cardiac affection is probably in some instances part of a general septicæmia, and yet in which there is no discoverable local lesion acting as a source of infection. I think there can be no doubt that such cases exist, and when they are associated with endocardial lesion it may become a matter of controversy whether the general state or the endocardial lesion is to

be regarded as the initial factor. The condition varies within certain limits, but it is always marked by symptoms of a very violent type which pass under the generic name of "blood-poisoning," and this is often the nearest approach to diagnosis which is attained. A fatal issue is not long delayed, and with our present knowledge we have to be satisfied with the diagnosis of the disease, and a prognosis without hope. The affection may attack a hitherto healthy heart, but may be superadded to an old valvular lesion, and supervene in the course of an acute illness. A good example of this latter is seen in the following case:—



Temperature Chart of Case XX.

CASE XX.—John G., æt. 26, puddler, was admitted into the Wolverhampton Hospital on the 17th of March, 1879. He presented the ordinary signs of pneumonia at the right apex, and of pleuro-pneumonia at the left base. The cardiac sounds were obscured, and no murmur was heard. The pulse was 120, the respirations 60, and the temperature 102°. For seven days he did very well, and gave us no special anxiety. The pulse varied from 120 to 130, the respirations from 30 to 40, and the temperature, as seen in the above chart, varied little. At night

occasionally there was delirium. He had diarrhœa, which proved most intractable, the bowels being moved from two to five times in twenty-four hours. There was a trace of albumen in the urine, which had, however, disappeared on the 20th. On the 23rd he perspired profusely, and the tongue was brown, dry, and glazed. On the 24th he continued to perspire profusely, and was delirious. The temperature rose nearly to 106°, the pulse to 144, and the urine contained albumen. On the 25th he was comatose, with contracted pupils. The temperature rose to 107°, the pulse to 160, and the bowels were moved seven times. This seemed a strange, and was certainly an unexpected, turn for the disease to take, and its real significance was much speculated on. At the *post-mortem* examination the intestines presented no trace of the lesions which characterise typhoid fever. The lungs revealed the accuracy of the diagnosis of the pulmonic condition. The left heart presented marked traces of old endocardial changes, the aortic valves being incompetent, and the mitral segments thickened. On the auricular surface of the aortic cusp of the mitral valve there was a cleanly-cut ulcer of about the size of a sixpenny piece, the cavity of which was filled with sanguineo-purulent looking matter, which, owing to an oversight, was not examined microscopically. The ulcer had almost perforated into the ventricle. In none of the other organs was there any change worthy of note. There can be no doubt that the ulcer on the endocardium was the cause of the increase in the severity of the patient's condition during the last few days of life. Its pathogenesis is, however, surrounded by difficulties. I do not incline to the opinion that the endocardial inflammation advanced hand in hand with the pulmonic disease, but that it was rather a later develop-

ment, and a complication of it. Whatever view be taken, the gravity of such a condition is apparent, and the possibility of its occurrence is to be taken into account when a change of such an unexpected and sudden nature supervenes in the course of an acute illness which, while displaying in individual cases considerable variety, yet runs a well defined course, and the dangers of which, are, as a rule, foreseen.

We now come to the consideration of that form of endocarditis, which, following Rosenstein, we designate vegetative or verrucose, and which holds an intermediate position between the two forms already considered.

The local condition is, we believe with Virchow, Ziegler, Cornil and Ranvier, Coats, and others, a true inflammation leading to a proliferation or infiltration of the deeper layer of the endocardium, and of the subendocardial tissue. This infiltration or proliferation forms, as seen from the interior of the heart, a *hillocky* swelling, over which the endocardium is intact. This is one stage, and resolution may take place at this point, or, the inflammatory process being stayed and resolution not occurring, earthy salts may be deposited at the seat of the inflammation. The hillocky swellings are, as a rule, small. Several may adjoin each other, but when this is the case there is usually a distinct furrow between the individual swellings. If from this stage the morbid process advances, the hillocky swellings become more prominent; and the endocardium, which had hitherto remained intact, begins to give way, largely owing to the pressure to which it is subjected from beneath, in a manner analogous to skin ulceration occurring over tumours. This is technically an ulceration, but it is an accidental, and secondary, and essentially a mechanical effect, the primary and the

important condition being the inflammation producing the tumefaction; for, as will be seen later, the condition makes itself known clinically before ulceration of the endocardium has occurred. No objection is here taken to the use of the term ulcerative so long as it is not used as a definition of the disease, but simply to express a mechanical result of it. To speak, however, of the disease as ulcerative endocarditis is misleading, and tends to perpetuate the confusion into which endocarditis, in its clinical aspects at least, has been allowed to fall. With the yielding and ulceration of the endocardium there is no escape of hitherto pent-up inflammatory or necrotic products into the circulation as in the true ulcerative form. All that escapes into the circulation in this case is the molecular detritus from the worn surface of the endocardium. The condition may be arrested at this stage also, and have its site occupied by lime salts, and it is even possible the endocardial wound may cicatrise. The process may, however, continue to advance, the new and ill-formed tissue still further raising the part above the surrounding surface, and leading more and more to the destruction of the endocardial covering, until it is only represented by a slightly raised ring round the base of the growth. No endocardium exists now between the adjoining hillocks; but the furrows, which originally marked their limits, may be still traceable, and help to give the mass that cauliflower-like appearance which, from the first, arrested the attention of observers.

That fibrin is usually or necessarily deposited on these cauliflower-like masses of vegetation is open to the gravest doubt. Did this take place, we should expect the spaces between what may be called the free fungating ends to be invariably occupied by the fibrinous deposit,

and that there would be a levelling up of the free surface of the mass until a raised and flat miniature plateau was produced; and we know that no such process as this takes place. Again, were the free points of such a mass to become the seat of fibrinous deposit, as is generally believed to be the case, pieces of it would be readily detached on the slightest cardiac disturbance, such as palpitation with its temporary increase of systolic power, and there would be a record of embolism far exceeding anything with which we meet. Although such a deposit is always described as part of the process, I believe that the majority of such "caps of fibrin" are developed during the stage of gradual failure of the heart, which often extends over a series of days before the end comes, and when, as is well known to clinical and pathological students, fibrinous deposits and coagula are invariably produced. If it were the case that fibrin is so readily deposited upon these vegetations as is assumed, we fail to see how the masses do not attain much greater dimensions, and why it is not as common to have masses of vegetations projecting half an inch from the endocardium as a quarter of an inch.

The free end of a vegetation occasionally gets detached, and produces symptoms depending upon the organ in which it is arrested, and which are too widely known to require consideration here.

As a rule the larger masses of vegetations do not undergo any curative process, but I am inclined to believe they occasionally do. Not infrequently we find on both the aortic and mitral valves large calcareous masses, which must have projected far into the blood stream, and it is difficult to explain these save on the assumption, that they were originally vegetative masses in which calcareous material had been deposited on the subsidence of the

inflammatory process. These calcareous masses are commonly covered with endocardium, and this appears to countenance the assumption that an abraded surface even on the endocardium may have its epithelium restored.

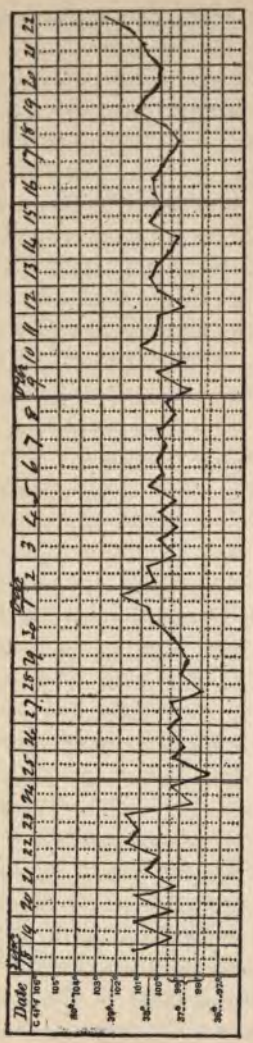
The relative frequency with which vegetations are found on the various parts of the heart have received such abundant attention, that it is unnecessary to consider this point here.

The clinical features of the affection are to be found in the following cases:—

Case XXI. was a gentleman who was under my care for a month during the year 1877, when *locum tenens* for a medical friend. He had been seen by physicians of eminence and ability, and none of those who saw him could throw any light on the clinical problem presented by his case. He was a young married man, and was known to have aortic disease of some years' standing. When under my care, and for some considerable time before, his condition was one of debility, associated with marked pyrexia and frequent pulse. The pyrexia was practically unaffected by the ordinary febrifuge remedies, as quinine, &c. The patient was nervous, and the circulation was readily excited. He had profuse perspirations, which became daily if not more frequent. There was nothing in the lungs to explain the phenomena, and typhoid fever was quite excluded. Albumen appeared in the urine before he passed from under my care. He became gradually worse, and finally died. A *post-mortem* examination was not allowed. The case left a deep impression on my mind because of its apparent obscurity. I hunted through such authorities on endocarditis as I had at my disposal, seeking for light, but found nothing to warrant me in believing that the case was of this

description. Some time afterwards I had the opportunity of studying the following case :—

CASE XXII.—Harriet A., æt. 31, married, was admitted into the Wolverhampton General Hospital on the 18th of September, 1878. She had had her first attack of rheumatic fever one year and eight months previously. On admission she suffered from considerable orthopnœa and palpitation. The pulse was 132, small, and irregular in force. The cardiac impulse was diffuse, its outer limit being visible in the fifth space in the nipple line. There was a systolic mitral murmur. Respiration was harsh over both lungs, but there were no râles. The liver was slightly enlarged. The bowels were constipated; the tongue watery and clean. The urine was neutral, its specific gravity 1018, and contained no albumen. There were no joint pains, and no œdema. For the first four days of her hospital life the morning temperature was about 99°, and the evening 101°. On the sixth day, the 23rd, there was some joint pain, and the morning temperature was 101°. She was put on 20 grains of salicylate of soda every four hours. The pain had disappeared, and the temperature was normal on the following morning, and as there was tinnitus aurium, the salicylate was stopped. During the day there was a tendency to delirium, which, however, soon passed off. For a week after this the temperature kept about 99°, and there was no return of the articular pain. On October 1st the temperature ran up to 102°, and continued about 100° till the 8th, being half a degree higher at night than in the morning; the pulse continued irregular, and was frequently intermitting. She had a nervous and startled aspect, and perspired profusely when asleep. On the 3rd there was a trace of albumen. From the 9th to the 23rd, when she died, her condition



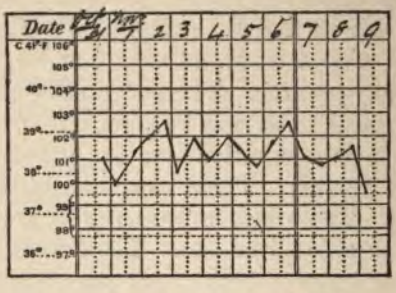
Temperature Chart of Case XXII.

became gradually worse. She became more restless and irritable, the slightest examination agitating her much; she was frequently delirious, and suffered from the terror of illusions. The pulse did not exceed 120. The temperature during this period presented a certain uniformity, ranging between 99° and 101°; it would rise from the former to the latter in twelve or twenty-four hours, and take from thirty-six to forty-eight hours to fall, making the chart line wave-like, each wave including three days. The evening before death the temperature rose to 102·6°, and the pulse to 132. On the 19th an aortic systolic murmur was heard for the first time. *Autopsy.*— There were universal pleuritic and pericardial adhesions of old date. The mitral orifice was slightly stenosed, its cusps being thickened. Surrounding the free margin of the valve, on its auricular aspect, there was a chain of vegetations, over which the endocardium was intact. On the under surface

of each segment of the aortic valve there was a chain of soft vegetations, most of which had lost their endocardial covering. Peyers patches were normal. The other organs presented no appearances worthy of note.

CASE XXIII.—Wm. J., æt. 35, cab-driver, was admitted into hospital on October 31st, 1878. He had had three attacks of rheumatic fever, the last attack having occurred thirteen years previous to the date of admission. Two months before admission he began to suffer from cough, shortness of breath, and pain across the chest. He used to drink heavily, but had been temperate for months. There was no history of strain. He was a well-built and muscular man with a florid complexion. The expression was anxious, his manner tremulous and agitated. He could lie in any position. The perspiration stood in beads on his face. The pulse was 116, regular, but collapsing. There was heaving pulsation over the greater part of the front of the thorax. There was considerable cardiac enlargement. There was a loud double aortic murmur, and a faint systolic mitral one. There were subcrepitant râles over the bases of both lungs posteriorly. The liver was enlarged but not tender. The appetite was poor; there was considerable thirst; and the bowels were constipated. The urine was acid, and its specific gravity was 1015, there was a large deposit of urates, and it contained albumen, but no casts were found. There was no joint pain and no œdema. The temperature was 101°. Up to the time of his death there was little variation in the condition. The tremulous and agitated murmur persisted; perspiration continued profuse; he slept much, both day and night, but was easily roused. He was never delirious; but always intelligent and rational. The pulse varied from 88 to 100, and became more typically aortic. The

respiration never exceeded 28. The temperature varied between 100° and 102·6°: it was higher in the evening than in the morning, except twice, when the contrary was the case. On the morning of the 9th of November, when sitting up to use the bed-pan, he fell from his bed and expired on the floor. *Autopsy.*—In addition to chronic sclerotic endocardial changes producing a degree of stenosis and incompetence at the left ostium venosum, and likewise incompetence of the aortic valves, there was a cauliflower-like mass of vegetations, about the size of a shilling, on the ventricular surface of the anterior segment

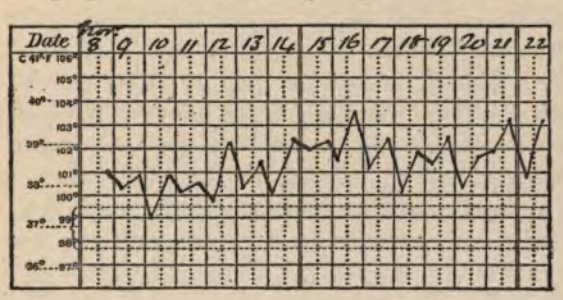


Temperature Chart of Case XXIII.

of the mitral valve, numerous smaller vegetations on the ventricular wall opposite to this, as well as some on the under surface of the aortic cusps. Over all these the endocardium was destroyed. The spleen contained a large infarct. There was slight cirrhosis of the kidneys. No embolism was found in the brain or in any other organ.

Case XXIV.—Frank P., æt. 15, confectioner's assistant, was admitted into hospital on 8th November, 1878. Three years previously he had had his first attack of rheumatic fever; twelve months previously he had his last attack, which was accompanied with pericarditis. He had the

same anxious appearance, and nervous and agitated manner as the two preceding cases had, but in him the nervous agitation was more extreme, and rendered thorough examination impossible. The pulse was 138, irregular, very small, and at times scarcely perceptible. The respirations were 64. There was a diffused undulatory impulse over the precordia, and the cardiac sounds were faint and accompanied by a systolic murmur at the apex. The temperature was 101°. The urine deposited abundant urates, but there was no albumen. The pulse was improved and the palpitation relieved by digitalis. He was kept



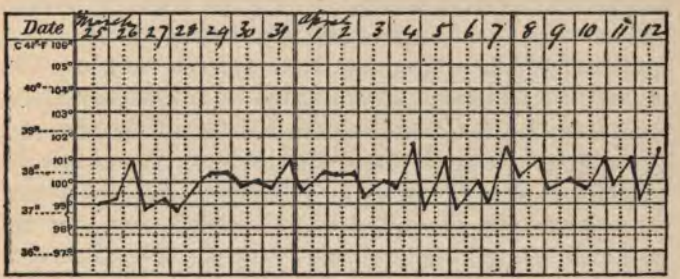
Temperature Chart of Case XXIV.

very quiet and not allowed to sit up in bed for any purpose. Until death, which occurred on the 23rd, he perspired frequently but not profusely; respirations varied from 48 to 58, and ran up to 66 before death. The pulse varied from 128 to 136, reaching 146 before death. The temperature oscillated between 100° and 103·4°, and was invariably higher at night than in the morning. *Autopsy.*—There were old pericardial adhesions. The heart was much enlarged and pushed the left lung upwards, the greater part of its lower lobe being collapsed. There was some fluid in both pleuræ. The left ostium venosum was

enlarged. On the under surface of each aortic cusp there was a mass of vegetations and another mass on the adjoining part of the endocardium.

CASE XXV.—Ellen E., æt. 15, was admitted into hospital on March 25th, 1879. Since the preceding Christmas she had suffered from palpitation and had gradually become weak and pale. Eleven months before admission there had been a slight appearance of the catamenia, but none since that date. She never had rheumatism, but her father had at the age of seventeen. She presented the ordinary appearances of anæmia; was of a happy disposition, and had no pain or discomfort during the time she was in hospital. The pulse was 116, small, and regular. The right external jugular vein was full and pulsating. There was slight pulsation over the precordia. The apex impulse was in the fifth space, $4\frac{1}{2}$ inches from midsternum and $1\frac{1}{4}$ in. outside the nipple line. There was a loud systolic murmur heard over the chest anteriorly and posteriorly; a sytolic murmur was also audible over the occiput, and there was a *bruit de diable* in the neck. The urine was acid, the sp. gr. 1016, and it contained a trace of albumen. The lungs were normal. The blood examined by the hemacytometer showed 69 per cent. of coloured corpuscles. For nearly three weeks there was nothing whatever to note in her condition, further than that the urine always contained a trace of albumen; that the apex murmur varied somewhat in tone and loudness; that the temperature ranged from 99° to 101·4°, and was almost always higher at night than in the morning. On the evening of April 12th she died suddenly and unexpectedly. On April 7th the corpuscles were 64 per cent. *Autopsy.*—There was extensive hæmorrhage into the membranes over the base of the brain, and

extending downwards over the spinal cord to the seventh cervical vertebra. There was a considerable clot around the medulla. No embolism or aneurism was found, but, from the distribution of the hæmorrhage it was probably due to embolism at the junction of the basilar and vertebral arteries; and that part of the vessels was unfortunately not removed with the brain. The heart weighed 11 ounces. The free margin of the mitral valve presented a fringe of vegetations, similar vegetations being on several of the musculi papillares as well as covering a large area on the anterior wall of the left auricle. Many



Temperature Chart of Case XXV.

of the vegetations had lost their endocardial covering. Two of the cordæ tendineæ were snapped, their free ends being calcareous. There was no fibroid thickening of the mitral segments. The aortic valves and the aorta were perfectly healthy. The kidneys were unusually large, weighing 11½ and 13 ounces respectively; but the enlargement was mainly due to their extraordinary length; the cortex in places was relatively increased and pale.

The following three cases I can only refer to briefly.

Case XXVI.—Wm. F., æt. 19, was under my care in Carlisle. He had a double aortic murmur, the result of

an attack of rheumatic fever from which he had suffered some years previously. During the time I attended him, in what proved to be his last illness, the features of his case may be briefly stated as follows:—elevated temperature, which was usually higher at night than in the morning, quick pulse, profuse perspirations, readily excited and irritable heart, and a nervous manner. There was no œdema, and no dyspnœa, although some acceleration of respiration existed. He died in a few weeks. At the *post-mortem* examination, in addition to old standing endocardial changes, there was a mass of vegetations, rather larger than a shilling, in the left ventricle, besides smaller vegetations on the aortic and mitral valves.

Case XXVII.—I can not do more than refer to. The patient was a friend of my own whom I did not see during the course of the illness, but the account of the case sent to me by the medical attendant so closely resembled the cases I had already seen, that I expressed the belief that the case was one of vegetative endocarditis, and that the prognosis was gloomy in the extreme. My fears only proved too true, for my friend died. At the *post-mortem* examination a large mass of vegetations was found in the left ventricle and several smaller masses. There had been no antecedent rheumatic fever, or cardiac lesion; but at the outset of the illness there had been some comparatively slight rheumatic pains.

Case XXVIII. occurred in the practice of my friend, Dr. Cormack Smith, of Gorebridge. The patient was a young woman who had had an attack of rheumatic fever, and although the joint pains had practically disappeared, with only occasional slight return in one or other joint, yet the temperature continued elevated, and the pulse was quickened. There were frequent perspirations, and

there was the same nervous, readily agitated manner, which has been referred to in some of the preceding cases. There was auscultatory evidence of valvular lesion. From my experience up to that time, the prognosis seemed very hopeless. My friend informed me that she died a comparatively short time after I saw her, and that dropsy had not developed.

From these eight cases it would appear that the form of endocarditis to which the term vegetative or verrucose is applied, is deserving of a place as a clinical entity; that its recognition is possible, and therefore desirable, both from the standpoint of scientific medicine, as well as from the duty we owe to our patients.

In reviewing them, we find that five (XXI., XXII., XXIII., XXIV., XXVI.) had had rheumatic fever at some former period, and one other (XXVII.) had had slight articular pains at the commencement of the illness; but none of them had any articular pain worthy of note during the time they were under observation. Case XXV. never had rheumatism of any kind, although her father, when a young man, had had rheumatic fever. Case XXVIII. was a continuation of an attack of rheumatic fever, although the patient had recovered from the articular affection, and was regarded as convalescent shortly before I saw her. Five (XXI., XXII., XXIII., XXIV., XXVI.) had previously produced valvular lesion. One (XXVII.) of the three others had no antecedent cardiac lesion; another (XXV.) probably had not; while in the other (XXVIII.) it dated from the immediately preceding rheumatic attack.

The clinical story may be outlined as follows:—A patient comes complaining of weakness which may have existed only for a short time, or may have extended over

a longer period, its course having been progressive, but slow. Palpitation may be complained of, and breathlessness on exertion. Perspirations may be referred to, and be regarded by the patient as bearing a causal relationship to the debility. The perspirations may be especially marked during sleep, and may only be present then. On inquiry, it is found that he has had one or more attacks of rheumatic fever; and he may be aware that, during one or other attack, his heart had been affected; but he may volunteer the information that he had recovered perfectly, and had been able to follow his occupation as before, but that he had of late felt more and more unfit for the daily round of duty. The pulse is found to be rapid, the agitation due to the examination sending it up to 120 to 130, or even more, but even after the subsidence of the agitation it maintains an accelerated rate of from 100 to 110 or more, although it may be as low as from 80 to 90. It may be fairly strong, and quite regular, but its characters depend much upon the valvular lesion present. The most superficial examination may reveal the presence of organic endocardial murmurs located at some of the cardiac orifices. There may be no dropsy, no swelling of the ankles, no pulmonary râles, indeed none of the ordinary signs of mechanical derangement of the circulation. The tongue may be clean, the bowels regular, the urine normal. There is no abdominal distension or tenderness. There is no splenic enlargement. The temperature is slightly elevated, perhaps only reaching 100°. This is often the picture first presented to us. The state of the tongue and the abdominal condition, combined with other factors, exclude the diagnosis of typhoid fever. The patient is confined to his room and kept under observation. The temperature is then found

to vary, and is at some period of the twenty-four hours normal. It may so happen that he has been seen for the first time by his medical attendant when the temperature was normal, and then, unless there is considerable familiarity with the various phases of the condition, it may easily be overlooked. In addition to the elevated temperature, the pulse, as a rule, keeps persistently accelerated. The manner becomes more nervous, the slightest excitement, such as the medical attendant's visit, producing considerable agitation, with increase of the pulse rate. So marked, indeed, has this been in some cases, that I have dreaded the possible effect of my visit. The perspirations continue, or become more marked. Later in the history, albumen may appear in the urine, and, in some cases œdema, or even a degree of general dropsy, although my own experience has been confined to cases where dropsy never became an element requiring treatment. Delirium may ensue, and the patient may lapse into a semi-comatose state, speedily ending in death. Or the fatal issue may be more sudden, and be due to cardiac failure, or to some irrecoverable disturbance of cardiac equilibrium, or to cerebral embolism. Embolism in other organs gives rise to the usual symptoms of such incidents, and do not require to be referred to in detail here. It suffices to repeat that the emboli do not become infective centres.

The foregoing is the story as it is to be read in a case associated with antecedent cardiac lesion. The fatal result is not due to the mechanical lesion of valves, for there may be none of the disturbances present which tend towards a fatal termination in purely mechanical imperfections. The diagnosis of the mechanical imperfection alone can, indeed, be but a poor consolation to the thoughtful physician standing face to face with such a

case, and seeking for the pathological equivalent of the clinical phenomena. Our aim has been to define the latter, and to find the former.

The disease, however, presents other phases. It occurs without any record, save the most trifling, of antecedent rheumatism, and none whatever of cardiac lesion. Coming on with slight rheumatic pains, its onset and progress, perhaps from valve to valve, may be followed by the stethoscope, but, on the other hand, it has been frequently noted that the condition has been found after death when there had been no auscultatory signs of valvular lesion during life; and then its diagnosis must be surrounded with extreme difficulty, and could not attain a more positive place than that of a tentative possibility. These cases must, however, be extremely rare; for it is difficult to believe that there can be such grave endocardial lesion without the valvular mechanism being affected. There may, as we all know, be great valvular and endocardial changes without a murmur being audible; but this aspect of affairs is the result of the degeneration of the cardiac muscle, and if degeneration has been recognised, the absence of murmur does not lead us to negative the probability of valvular lesion. In the same way, if vegetations be associated with degeneration of muscle, there may be no murmur audible, no matter to what extent the valves may be affected; but even in these cases the diagnosis is attainable, although it may be difficult.

In such an instance as Case XXV., the question arose whether the mitral murmur might not be due to the anæmic condition. The elevated temperature, and the presence of a trace of albumen in the urine, warranted the diagnosis, although it was with great reluctance we yielded to the pressure of facts. The albumen might have been

regarded as a concomitant of the anæmia, but the temperature could not be so regarded. In such a case, however, the diagnosis can only be made by carefully weighing the various factors comprising it; and the importance of accuracy is forcibly brought out by the result of the case. Had we allowed the patient go about, instead of keeping her in bed, we should have blamed ourselves.

Another and a common phase is to be seen in the convalescent from acute rheumatism. The pains have probably disappeared, tending only to return slightly in one or other joint, and are readily checked by salicine or a salicyl salt. The pulse, however, continues accelerated, the temperature more or less elevated at some period of the twenty-four hours, and there are probably perspirations. There is probably endocardial murmur, which was noted early in the attack. In such a case, the problem before the physician is, What are the conditions leading to the phenomena? Are they to be regarded as the outcome of the rheumatic state, and simply as an expression of the presence of the rheumatic poison; or is their cause to be found in an anatomical instead of a chemical condition? I believe the phenomena are to be explained by the morbid changes taking place in the endocardium.

The presence of albumen in the urine is probably to be explained in many cases by the molecular *débris* from the broken endocardium finding a lodgment in the kidney; for the only case in which albumen was not present was in Case XXIV., and in it the endocardium had not given way over the vegetations. The delirium also may—at least, sometimes—be accounted for in this way, the *débris* becoming arrested in cerebral capillaries. Murchison (*Lancet*, 3rd May, 1879) has pointed out that there may be endocarditis with pyrexia, in which no emboli are found; but

it would require immense labour and care to positively assert that minute emboli were not present, and Virchow has been successful in finding them.

There is no doubt that isolated individuals have the power of recognising the clinical phenomena which indicate the presence of acute endocardial inflammation, but the knowledge necessary to its recognition is not general, and our text-books give us little assistance, and may indeed mislead. There is also, as has been already indicated, an undesirable confusion of terms and an insufficient differentiation of the various forms of endocardial inflammation. As illustrating the clinical side of this contention I noted when first studying this subject in 1879, a case recorded in one of the medical journals, which was as follows:—A mild case of chorea, where repeated examination revealed no cardiac murmur, and in which death took place from cerebral hæmorrhage, due to an embolus washed from endocardial vegetations. The temperature ranged from 98° to 101°; the patient looked "too ill for her symptoms," and there was a trace of albumen in the urine. The endocardial condition was not diagnosed even if suspected. Somewhat similar cases are to be found abundantly in the pages of current medical literature. They are not diagnosed, and not unfrequently the patient's death seems to surprise his attendant. I make bold to believe that, with our present knowledge, the diagnosis can be made; and that in the case referred to above, we should have regarded the diagnosis as attainable, even although there was no murmur, seeing that the case was the sequel to an attack of chorea, and occurred in a child, where the rapidity and brevity of the cardiac action tend to obscure murmurs, and that there was a high temperature, and albumen in the urine, and

what Walshe called the ".general aspect of acute illness." Doubtless the diagnosis is largely attained by a process of exclusion, but all methods belong to the true physician.

The preceding series of cases, presenting an unbroken line of mortality, led me at one time to form an utterly hopeless prognosis of this affection. A more extended experience has, however, led me to take a less hopeless view of their possible future, and it may be well to record briefly a few cases illustrating this side of the subject.

CASE XXIX.—A young woman, a patient of Dr. Lockie of Carlisle, was seen by me several times. She had had rheumatic fever, and antecedent cardiac lesion. The temperature was high, the pulse rapid, there were profuse perspirations, and endocardial murmurs were present. She was ill for weeks, but had no articular pains, and no œdema. Her condition gave great anxiety, but she ultimately recovered, as far as a person with organically altered valves can recover, and was able to go about as usual.

CASE XXX.—Margaret V—— consulted me on 4th January, 1881, complaining of weakness and swelled ankles. Three weeks before she had been treated for a cold. The legs below the knee were œdematous. The pulse was 100, and regular. The temperature in the afternoon was 100°. The temperature at night was 100·2°, the pulse 100. There was no cough, no diarrhœa, no abdominal tenderness or distension. The cardiac dulness extended slightly beyond the nipple line, and at the apex there was a systolic murmur propagated towards the axilla; the pulmonary second sound was accentuated. There was no anæmia, and the girl was well nourished. She had never had rheumatism, but some of her near relatives were rheumatic. There was no albumen in the

urine. On 6th January the temperature was 100° and the pulse 108, and she had perspired occasionally since my last visit. The œdema had lessened. A blister to the præcordia, and 5 grs. of iodide of potassium, to be taken three times a-day, were ordered. On *9th January*, the temperature was 99°, and the pulse 100. The œdema had disappeared. On *13th January*, the temperature was 99° and the pulse 96. There was no trace of the murmur discoverable. On *20th January*, the evening temperature was normal, and the pulse was 80. The iodide was stopped, and iron was ordered. The murmur had not reappeared.

CASE XXXI. was a young man about thirty, a patient of Dr. Lockie of Carlisle whom I had the opportunity of seeing with Dr. Lockie, and afterwards for him for some time. He was convalescent from acute rheumatism in so far that the joint pains had been subdued, although they tended to return in one or other joint. The temperature kept up, and the pulse was quickened, and there were frequent perspirations. An interesting observation made by Dr. Lockie, and pointed out to me, was that the systolic murmur at the apex was only audible when the patient lay on the right side with an inclination to the prone position. The condition persisted for several weeks, but he ultimately recovered, with no trace of cardiac lesion.

I do not think there is any room for doubt as to the diagnosis in the last three cases. But it becomes an interesting question as to what influence the treatment had in bringing about a favourable result. The treatment in them was quite different from that followed in my other cases, all of which had proved fatal, and was confined to blistering of the præcordia with Spanish fly, and the administration of iodide of potassium internally, with

salicine for the relief of articular pain when necessary. With reference to the beneficial effects to be derived from blisters, applied locally, I may say that I have had the opportunity of witnessing the marvellously sedative and beneficial effect of issues applied to the præcordia by a physician for whom I have profound respect, but who had been educated in an older school than I. From the results which I have witnessed I believe we have in blisters, and probably in issues, remedial agents which are not to be discarded, even in endocardial inflammations, however obscure the rationale of such treatment may be. Iodide of potassium is administered on the well-recognised principle, that it aids in the absorption of inflammatory products, and also allays, I believe, the cardiac irritability. Digitalis I not only regard with little favour, but believe to be absolutely dangerous in this condition. In my earlier cases I used it, and was much dissatisfied with its action. It did not, as a rule, reduce the rapidity or increase the power of the heart; on the contrary, it, as a rule, seemed to increase the irritability both of the heart and of the patient. On theoretical grounds it is also to be condemned, for an increase in systolic power might readily lead to the detaching of a portion of vegetation which, if arrested in a cerebral vessel, might be speedily fatal. This effect I have witnessed, and believe the deduction legitimate that digitalis played a part in the process.

One other form of vegetation it seems desirable to mention before leaving the subject. In the hearts of persons dying from certain acute diseases, of a febrile type, there are not unfrequently present small masses of fibrin of a sago-like appearance, which are adherent to the endocardium, and can readily be separated by the nail,

leaving the endocardium upon which they rested practically intact. Around the base of these concretions there may be seen one or more minute vascular twigs. The last occasion on which I noted this condition was in a young woman, the subject of old standing cardiac disease, who, a few days after being delivered of twins, died of what is clinically known as puerperal tetanus, and which was associated with a very high temperature. These concretions may also be found attached to spots of endocardium, which seem to have been the seat of a former inflammation, or of a degenerative process, and which, on close examination, appear of a whiter colour than the surrounding endocardium. In the former, the concretions no doubt rest on an inflamed spot of endocardium, in the latter, on a part the vitality of which has been diminished. In both cases they are formed in the days or hours preceding death, when the heart is, if we may use the term, blood-logged.

The pathogenesis of the disease we have considered is included in the larger question as to the nature of the morbid process in acute rheumatism, and what element in the blood so frequently lights up inflammation.

Balthazar Foster's interesting observations on the effect of lactic acid in producing all the symptoms of acute rheumatism have not been accepted as conclusive, although they are suggestive when taken in connection with the experimental production of endocarditis in animals by the injection of lactic acid (Dr. B. W. Richardson's *Experimental Inquiry on Endocarditis*). It has, however, to be borne in mind that endocarditis may accompany or follow other febrile affections—such as measles, scarlatina, &c.—where the poison is probably of quite a different type.

Dr. Goodhart's suggestion, that "ulcerative" endocarditis is due to epidemic influences, is of special interest at the present time, when the standpoint from which disease has been hitherto regarded is being rapidly transferred to one which, it is believed, will be of greater vantage. At the same time, it is necessary to accept with caution any inference drawn from the coincidence of two or three cases of any kind being under observation at the same time, for in practice it is frequently noted that not only rare cases, but births and deaths, tend to occur in groups.

The questions, therefore, remain: What is the nature of the rheumatic process? What element determines or lights up the local inflammations? Is the determining agent chemical or vital?

If we regard acute rheumatism as the expression of a faulty chemistry, determined by hereditary, or by less remote circumstances, or by a combination of these, we shall, I take it, be compelled to attribute the first step to it. On this hypothesis, valves would be irritated and inflamed by the circulation through them of a fluid containing a chemical irritant, and I presume no one is prepared to question the fact that chemical irritants may act thus.

The structure of the valves, and the manner in which they are nourished, must expose them in a special manner to the action of irritants. The fluid which nourishes them is slowly changed, and when it contains any irritating principle, its lingering contact with the tissues will afford exceptional opportunities for the production of a degree of irritation which readily becomes an inflammation. It is probable that the lowered power of the heart in febrile affections, by diminishing the pressure exercised during systole on the valves, as on the rest of the endocardial

surface, is an important factor in allowing an abnormal sluggishness in the nutritive stream, and if, as has been said, the nutritive fluid is abnormally irritating, its facilities for exciting inflammation are thus greatly aided.

In a considerable proportion of cases micrococci have been found in endocardial vegetations, and some ascribe the pathogenesis of the affection to them. In the present state of our knowledge the relation of these organisms is not altogether apparent. In a certain class of case where the cardiac lesion is but part of a septic condition, and secondary, there can be no doubt that the micrococci constitute an integral part of the local cardiac lesion, but then the cardiac lesion is a coincidence which is by no means constant, for many cases of septicæmia and pyæmia present no endocardial inflammation. Professor Ogston first pointed out the presence of micrococci in acute abscesses, and accepting this as an established fact, verified by Mr. Watson Cheyne and others, we are forced to conclude that there may be micrococci in the blood, either normally, or occasionally and accidentally, which, coming in contact with a part, the nutrition of which may be lowered or specially modified, may set up active local changes, or may materially modify the nature of the local changes already initiated. This seems to me as far as we can at present go, and much work has to be done before we can attain a more precise and satisfactory understanding of the relations of these organisms in this and analogous cases. Meanwhile, it is desirable that treatment should not be influenced too much by theoretical problems, but that we should in the main be guided by a tested empiricism.

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what Walshe called the ".general aspect of acute illness." Doubtless the diagnosis is largely attained by a process of exclusion, but all methods belong to the true physician.

The preceding series of cases, presenting an unbroken line of mortality, led me at one time to form an utterly hopeless prognosis of this affection. A more extended experience has, however, led me to take a less hopeless view of their possible future, and it may be well to record briefly a few cases illustrating this side of the subject.

CASE XXIX.—A young woman, a patient of Dr. Lockie of Carlisle, was seen by me several times. She had had rheumatic fever, and antecedent cardiac lesion. The temperature was high, the pulse rapid, there were profuse perspirations, and endocardial murmurs were present. She was ill for weeks, but had no articular pains, and no œdema. Her condition gave great anxiety, but she ultimately recovered, as far as a person with organically altered valves can recover, and was able to go about as usual.

CASE XXX.—Margaret V—— consulted me on 4th January, 1881, complaining of weakness and swelled ankles. Three weeks before she had been treated for a cold. The legs below the knee were œdematous. The pulse was 100, and regular. The temperature in the afternoon was 100°. The temperature at night was 100·2°, the pulse 100. There was no cough, no diarrhœa, no abdominal tenderness or distension. The cardiac dulness extended slightly beyond the nipple line, and at the apex there was a systolic murmur propagated towards the axilla; the pulmonary second sound was accentuated. There was no anæmia, and the girl was well nourished. She had never had rheumatism, but some of her near relatives were rheumatic. There was no albumen in the